

following accomplishment of the requirements of this paragraph.

(1) For airplanes on which the flight control computers (FCC) have not been modified in accordance with the requirements of paragraph (b) of this AD:

"Overriding the autopilot (AP) in pitch axis does not cancel the AP autotrim when LAND TRACK mode [green LAND on both Flight Mode Annunciators (FMA)] or GO-AROUND mode is engaged. In these modes, if the pilot counteracts the AP, the autotrim will trim against pilot input. This could lead to a severe out-of-trim situation in a critical phase of flight."

(2) For airplanes on which the FCC's have been modified in accordance with requirements of paragraph (b) of this AD.

"Overriding the autopilot (AP) in pitch axis does not cancel the AP autotrim when LAND TRACK mode (green LAND on both FMA's) is engaged, or GO-AROUND mode is engaged below 400 feet radio altitude (RA). In these modes, if the pilot counteracts the AP, the autotrim will trim against pilot input. This could lead to a severe out-of-trim situation in a critical phase of flight."

(b) For airplanes equipped with FCC's having either part number (P/N) B470ABM1 (for Model A310 series airplanes) or B470AAM1 (for Model A300-600 series airplanes): Within 60 days after November 2, 1994 (the effective date of AD 94-21-07, amendment 39-9049), modify the FCC's in accordance with Airbus Service Bulletin A310-22-2036, dated December 14, 1993 (for Model A310 series airplanes), or Airbus Service Bulletin A300-22-6021, Revision 1, dated December 24, 1993 (for Model A300-600 series airplanes), as applicable.

Note 2: Paragraph (b) of this AD merely restates the requirements of paragraph (b) of AD 94-21-07, amendment 39-9049. As allowed by the phrase, "unless accomplished previously," specified in the compliance statement of this AD, if those requirements of AD 94-24-07 have already been accomplished, this AD does not require that those actions be repeated.

(c) As of November 2, 1994 (the effective date of AD 94-21-07, amendment 39-9049), no person shall install an FCC having either P/N B470ABM1 or B470AAM1 on any airplane.

(d) An alternative method of compliance or adjustment of the compliance time that provides an acceptable level of safety may be used if approved by the Manager, Standardization Branch, ANM-113, FAA, Transport Airplane Directorate. Operators shall submit their requests through an appropriate FAA Principal Maintenance Inspector, who may add comments and then send it to the Manager, Standardization Branch, ANM-113.

Note 3: Information concerning the existence of approved alternative methods of compliance with this AD, if any, may be obtained from the Standardization Branch, ANM-113.

(e) Special flight permits may be issued in accordance with sections 21.197 and 21.199 of the Federal Aviation Regulations (14 CFR 21.197 and 21.199) to operate the airplane to a location where the requirements of this AD can be accomplished.

Issued in Renton, Washington, on October 4, 1995.

Darrell M. Pederson,

Acting Manager, Transport Airplane Directorate, Aircraft Certification Service.

[FR Doc. 95-25161 Filed 10-10-95; 8:45 am]

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DEPARTMENT OF ENERGY

Federal Energy Regulatory Commission

18 CFR Part 35

[Docket No. RM95-8-000]

Promoting Wholesale Competition Through Open Access Non-Discriminatory Transmission Services by Public Utilities; Notice of Potential Broadcast of Technical Conferences

October 4, 1995.

AGENCY: Federal Energy Regulatory Commission, DOE.

ACTION: Notice of Potential Broadcast of Technical Conferences.

SUMMARY: The Federal Energy Regulatory Commission is notifying persons interested in the Commission's technical conferences in the captioned proceeding of the opportunity, for a fee, to receive the broadcast of the conferences. This notice provides interested persons with the necessary information by which they may seek to receive the broadcast of the conferences.

DATES: Persons interested in the broadcast of the conferences must notify Julia Morelli or Shirley Al-Jarani at the Capitol Connection (703-993-3100) by October 12, 1995.

FOR FURTHER INFORMATION CONTACT:

Richard Armstrong, Office of Electric Power Regulation, 825 North Capitol St., N.E., Washington, D.C. 20426, (202) 208-0241, (fax) (202) 208-0180
Lawrence Anderson, Office of Electric Power Regulation, 825 North Capitol Street, N.E., Washington, D.C. 20426, (202) 208-0575, (fax) (202) 208-0180

SUPPLEMENTARY INFORMATION: In addition to publishing the full text of this document in the **Federal Register**, the Commission also provides all interested persons an opportunity to inspect or copy the contents of this document during normal business hours in Room 3104, at 941 North Capitol Street, N.E., Washington, D.C. 20426.

The Commission Issuance Posting System (CIPS), an electronic bulletin board service, provides access to the texts of formal documents issued by the Commission. CIPS is available at no charge to the user and may be accessed

using a personal computer with a modem by dialing (800) 856-3920. To access CIPS, set your communications software to 19200, 14400, 12000, 9600, 7200, 4800, 2400 or 1200bps, full duplex, no parity, 8 data bits and 1 stop bit. The full text of this document will be available on CIPS in ASCII and WordPerfect 5.1 format. The complete text on diskette in WordPerfect format may also be purchased from the Commission's copy contractor, La Dorn Systems Corporation, also located in Room 3104, 941 North Capitol Street, N.E., Washington, D.C. 20426.

Please take notice that, for a fee, the Capitol Connection may broadcast technical conferences in this proceeding to interested persons. These technical conferences are: ¹ (a) October 26, 1995—Commission technical conference on ancillary services; (b) October 27, 1995—staff conference on *pro forma* tariffs; (c) December 5 and 6, 1995—Commission technical conference on comparability for power pools. Persons interested in receiving such broadcasts should contact Julia Morelli or Shirley Al-Jarani at the Capitol Connection (703-993-3100) no later than October 12, 1995.

Lois D. Cashell,

Secretary.

[FR Doc. 95-25170 Filed 10-10-95; 8:45 am]

BILLING CODE 6717-01-M

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 50

[AD-FRL-5313-4]

RIN 2060-AC06

National Ambient Air Quality Standards for Nitrogen Dioxide: Proposed Decision

AGENCY: Environmental Protection Agency (EPA).

ACTION: Proposed decision.

SUMMARY: The level for both the existing primary and secondary national ambient air quality standards (NAAQS) for nitrogen dioxide (NO₂) is 0.053 parts per million (ppm) (100 micrograms per meter cubed (µg/m³)) annual arithmetic average. In accordance with the provisions of sections 108 and 109 of the Clean Air Act (Act), as amended, the EPA has conducted a review of the criteria upon which the existing NAAQS for NO₂ are based. The revised

¹ The time and place of the technical conferences was provided in an earlier notice, issued August 17, 1995. 60 FR 43997 (August 24, 1995).

criteria are being published simultaneously with the issuance of this proposed decision. After evaluating the revised health and welfare criteria, under section 109(d)(1) of the Act, the Administrator has determined that it is not appropriate to propose any revisions to the primary and secondary NAAQS for NO₂ at this time.

DATES: *Comments.* Written comments on this proposal must be received on or before January 9, 1996.

Public Hearing. Persons wishing to present oral testimony pertaining to this proposal should contact EPA at the address below by October 26, 1995. If anyone contacts EPA requesting to speak at a public hearing, a separate notice will be published announcing the date, time, and place where the hearing will be held.

ADDRESSES: Comments on this proposed action should be sent in duplicate to: U.S. Environmental Protection Agency, Air and Radiation Docket and Information Center (6102), Room M-1500, 401 M Street, SW, Washington, DC 20460, ATTN: Docket No. A-93-06. The docket, which contains materials relevant to this proposed decision, is available for public inspection and copying (a reasonable fee may be charged) weekdays between 8:00 a.m. and 5:30 p.m. in the Central Docket Section (CDS) of EPA, South Conference Center, Room M-1500, telephone (202) 260-7548.

Public Hearing. Persons wishing to present oral testimony pertaining to this proposal should notify Ms. Chebryll C. Edwards, U.S. Environmental Protection Agency, Office of Air Quality Planning Standards, Air Quality Strategies and Standards Division, Health Effects and Standards Group (MD-15), Research Triangle Park, NC 27711, telephone number (919) 541-5428.

FOR FURTHER INFORMATION CONTACT: Ms. Chebryll C. Edwards, U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, Air Quality Strategies and Standards Division (MD-15), Research Triangle Park, NC 27711, telephone (919) 541-5428.

SUPPLEMENTARY INFORMATION: Availability of Related Information. The revised criteria document, "Air Quality Criteria for Oxides of Nitrogen" (three volumes, EPA-600/8-91/049aF-cF, August 1993: Volume I, NTIS #PB95124533, \$52.00; Volume II, NTIS #PB124525, \$77.00; Volume III, NTIS #PB95124517, \$77.00), and the final revised OAQPS Staff Paper, "Review of the National Ambient Air Quality Standards for Nitrogen Oxides: Assessment of Scientific and Technical

Information," (EPA-452/R-95-005, September 1995) are available from: U.S. Department of Commerce, National Technical Information Service, 5285 Port Royal Road, Springfield, Virginia 22161, or call 1-800-553-6847 (a handling charge will be added to each order). Other documents generated in connection with this standard review, such as air quality analyses and relevant scientific literature, are available in the EPA docket identified above.

The contents of this action are listed in the following outline:

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I. Background

A. Legislative Requirements

1. The Standards

Two sections of the Act govern the establishment and revision of NAAQS. Section 108 (42 U.S.C. 7408) directs the Administrator to identify pollutants which "may reasonably be anticipated to endanger public health and welfare" and to issue air quality criteria for them. These air quality criteria are to "accurately reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health or welfare which may be expected from the presence of [a] pollutant in the ambient air * * *."

Section 109 (42 U.S.C. 7409) directs the Administrator to propose and promulgate "primary" and "secondary" NAAQS for pollutants identified under section 108. Section 109(b)(1) defines a primary standard as one "the attainment and maintenance of which, in the judgment of the Administrator, based on the criteria and allowing an adequate margin of safety, (is) requisite to protect the public health." A secondary standard, as defined in section 109(b)(2), must "specify a level of air quality the attainment and maintenance of which, in the judgment of the Administrator, based on (the) criteria, is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of (the) pollutant in the ambient air." Welfare effects as defined in section 302(h) (42 U.S.C. 7602(h)) include, but are not limited to, "effects on soils, water, crops, vegetation, manmade materials, animals, wildlife, weather, visibility and climate, damage to and deterioration of property, and hazards to transportation, as well as effects on economic values and on personal comfort and well-being."

The U.S. Court of Appeals for the District of Columbia Circuit has held that the requirement for an adequate margin of safety for primary standards was intended to address uncertainties associated with inconclusive scientific and technical information available at the time of standard setting. It was also intended to provide a reasonable degree of protection against hazards that research has not yet identified (*Lead Industries Association v. EPA*, 647 F.2d 1130, 1154 (D.C. Cir. 1980), *cert. denied*, 101 S. Ct. 621 (1980); *American Petroleum Institute v. Costle*, 665 F.2d 1176, 1177 (D.C. Cir. 1981), *cert. denied*, 102 S. Ct. 1737 (1982)). Both kinds of uncertainties are components of the risk associated with pollution at levels below those at which human health effects can be said to occur with reasonable scientific certainty. Thus, by selecting primary standards that provide an adequate margin of safety, the Administrator is seeking not only to prevent pollution levels that have been demonstrated to be harmful but also to prevent lower pollutant levels that may pose an unacceptable risk of harm, even if the risk is not precisely identified as to nature or degree.

In selecting a margin of safety, the EPA considers such factors as the nature and severity of the health effects involved, the size of the sensitive population(s) at risk, and the kind and degree of the uncertainties that must be addressed. Given that the "margin of safety" requirement by definition only

comes into play where no conclusive showing of adverse effects exists, such factors, which involve unknown or only partially quantified risks, have their inherent limits as guides to action. The selection of any numerical value to provide an adequate margin of safety is a policy choice left specifically to the Administrator's judgment (*Lead Industries Association v. EPA, supra*, 647 F.2d at 1161-62).

Section 109(d)(1) of the Act requires that "not later than December 31, 1980, and at 5-year intervals thereafter, the Administrator shall complete a thorough review of the criteria published under section 108 and the national ambient air quality standards * * * and shall make such revisions in such criteria and standards * * * as may be appropriate * * *." Section 109(d)(2) (A) and (B) requires that a scientific review committee be appointed and provides that the committee "shall complete a review of the criteria * * * and the national primary and secondary ambient air quality standards * * * and shall recommend to the Administrator any * * * revisions of existing criteria and standards as may be appropriate * * *."

The process by which the EPA has reviewed the existing air quality criteria and standards for NO₂ under section 109(d) is described later in this notice.

2. Related Control Requirements

States are primarily responsible for ensuring attainment and maintenance of ambient air quality standards. Under title I of the Act (42 U.S.C. 7410), States are to submit, for EPA approval, State implementation plans (SIP's) that provide for the attainment and maintenance of such standards through control programs directed to sources of the pollutants involved. The States, in conjunction with the EPA, also administer the prevention of significant deterioration program (42 U.S.C. 7470-7479) for these pollutants. In addition, Federal programs provide for nationwide reductions in emissions of these and other air pollutants through the Federal Motor Vehicle Control Program under title II of the Act (42 U.S.C. 7521-7574), which involves controls for automobile, truck, bus, motorcycle, and aircraft emissions; the new source performance standards under section 111 (42 U.S.C. 7411); and the national emission standards for hazardous air pollutants under section 112 (42 U.S.C. 7412).

B. Existing Standards for Nitrogen Dioxide

The principal focus of this standard review is the health and welfare effects associated with exposure to NO₂ and other oxides of nitrogen. Nitrogen dioxide is a brownish, highly reactive gas which is formed in the ambient air through the oxidation of nitric oxide (NO). Nitrogen oxides (NO_x), the term used to describe the sum of NO and NO₂, play a major role in the formation of ozone in the atmosphere through a complex series of reactions with volatile organic compounds. A variety of NO_x compounds and their transformation products occur both naturally and as a result of human activities.

Anthropogenic (i.e., man-made) sources of NO_x emissions account for a large majority of all nitrogen inputs to the environment. The major sources of anthropogenic NO_x emissions are mobile sources and electric utilities. Ammonia and other nitrogen compounds produced naturally do play a role in the cycling of nitrogen through the ecosystem.

At elevated concentrations, NO₂ can adversely affect human health, vegetation, materials, and visibility. Nitrogen oxide compounds also contribute to increased rates of acidic deposition. Typical peak annual average ambient concentrations of NO₂ range from 0.007 to 0.061 ppm ("Air Quality Criteria for Oxides of Nitrogen," (Criteria Document or CD), U.S. EPA, 1993, p. 7-10). The highest hourly NO₂ average concentrations range from 0.04 to 0.54 ppm (CD, 1993, p. 7-10). Currently, all areas of the U.S., including Los Angeles (which is the only area to record violations in the last decade), are in attainment of the annual NO₂ NAAQS of 0.053 ppm. The origins, concentrations, and effects of NO₂ are discussed in detail in the "Review of National Ambient Air Quality Standards for Nitrogen Dioxide: Assessment of Scientific and Technical Information," (Staff Paper or SP) (SP, U.S. EPA, 1995) and in the revised Criteria Document (CD, 1993).

On April 30, 1971, under section 109 of the Act, EPA promulgated identical primary and secondary NAAQS for NO₂ at 0.053 ppm annual average (36 FR 8186). The scientific and medical bases for these standards are contained in the original criteria document, "Air Quality Criteria for Nitrogen Oxides," (CD, 1971).

On December 12, 1978 (43 FR 58117), the EPA announced the first review and update of the 1971 NO₂ criteria in accordance with section 109(d)(1) of the Act as amended. In preparing the Air

Quality Criteria Document, the EPA provided a number of opportunities for external review and comment. The Clean Air Scientific Advisory Committee (CASAC) of the EPA Science Advisory Board held meetings in 1979 and 1980 before providing written closure on the revised criteria document in June 1981 (Friedlander, 1981). This process resulted in the production of the revised 1982 document, "Air Quality Criteria for Oxides of Nitrogen" (U.S. EPA, 1982a).

A staff paper, which identified critical issues and summarized staff interpretation of key studies, received verbal closure at a CASAC meeting in November 1981 and formal written closure in July 1982 (Friedlander, 1982). In the Staff Paper (U.S. EPA, 1982), staff recommended that the Administrator select an annual standard "at some level between 0.05 ppm and 0.08 ppm." Based on the analysis of the criteria, staff concluded that choosing an annual standard within this range would "provide a reasonable level of protection against potential short-term peaks."

On February 23, 1984, the EPA proposed to retain both the annual primary and secondary standards at 0.053 ppm annual average and to defer action on the possible need for a separate short-term primary standard until further research on health effects of acute exposures to NO₂ could be conducted (49 FR 6866). The CASAC met to consider the Agency's proposal on July 19-20, 1984. In an October 18, 1984 closure letter based on weight of evidence, CASAC concurred with the Agency's recommendation to retain the annual average primary and secondary standards at 0.053 ppm (Lippmann, 1984). The CASAC further concluded that, "while short-term effects from nitrogen dioxide are documented in the scientific literature, the available information was insufficient to provide an adequate scientific basis for establishing any specific short-term standard * * *." After taking into account public comments, the final decision to retain the NAAQS for NO₂ was published by EPA in the **Federal Register** on June 19, 1985 (50 FR 25532).

C. Review of Air Quality Criteria and Standards for Oxides of Nitrogen

On July 22, 1987, in response to requirements of section 109(d) of the Act, the EPA announced that it was undertaking plans to revise the 1982 Air Quality Criteria Document for Oxides of Nitrogen (52 FR 27580). The EPA held public workshops in July 1990 to evaluate the scientific data being considered for integration into the CD.

In November 1991, the EPA released the revised CD for public review and comment (56 FR 59285).

The revised CD provides a comprehensive assessment of the available scientific and technical information on health and welfare effects associated with NO₂ and NO_x. The CASAC reviewed the CD at a meeting held on July 1, 1993 and concluded in a closure letter to the Administrator that the CD “* * * provides a scientifically balanced and defensible summary of current knowledge of the effects of this pollutant and provides an adequate basis for EPA to make a decision as to the appropriate NAAQS for NO₂” (Wolff, 1993).

In the summer of 1995, the Office of Air Quality Planning and Standards (OAQPS) finalized the document entitled, “Review of the National Ambient Air Quality Standards for Nitrogen Dioxide: Assessment of Scientific and Technical Information,” (SP, U.S. EPA, 1995). The Staff Paper summarizes and integrates the key studies and scientific evidence contained in the revised CD and identifies the critical elements to be considered in the review of the NO₂ NAAQS.

The Staff Paper received external review at a December 12, 1994 CASAC meeting. The CASAC comments and recommendations were reviewed by EPA staff and incorporated into the final draft of the Staff Paper as appropriate. The CASAC reviewed the final draft of the Staff Paper in June 1995 and responded by written closure letter (see docket A-93-06).

D. Decision Docket

In 1993, the EPA created a docket (Docket No. A-93-06) for this proposed decision. This docket incorporates by reference a separate docket established for the criteria document revision (Docket No. ECAO-CD-86-082).

E. Litigation

On July 21, 1993, the Oregon Natural Resources Council and Jan Nelson filed suit under section 304 of the Act to compel the EPA to complete its periodic review of the criteria and standards for NO₂ under section 109(d)(1) of the Act (*Oregon Natural Resources Council v. Carol M. Browner*, No. 91-6529-HO (D.Or.)). The plaintiffs and the EPA agreed to a consent decree establishing a schedule for review of the NO₂ NAAQS, which was subsequently modified pursuant to a further agreement between the parties. The U.S. District Court for the District of Oregon entered an order on February 8, 1995

requiring the EPA Administrator to publish a **Federal Register** notice announcing her decision on whether or not to propose any modification of the NAAQS for NO₂ by October 2, 1995. The order also requires the Administrator to sign a notice to be published in the **Federal Register** announcing the final decision whether or not to modify the NO₂ NAAQS by October 1, 1996.

II. Rationale for Proposed Decision

A. The Primary Standard

1. Basis for the Existing Standard

The current primary NAAQS for NO₂ is 0.053 ppm (100 µg/m³), averaged over 1 year. In selecting the level for the current standard, the Administrator made judgments regarding the lowest reported effect levels, sensitive populations, nature and severity of health effects, and margin of safety. After assessing the evidence, the Administrator concluded that the annual standard of 0.053 ppm adequately protected against adverse health effects associated with long-term exposures and provided some measure of protection against possible short-term health effects. The June 19, 1985 **Federal Register** notice (50 FR 25532) provides a detailed discussion of the bases for the existing standard.

2. Proposed Decision on the Primary Standard

The Administrator has determined that it is not appropriate to propose any revisions of the existing NO₂ primary standard at this time. In reaching this proposed decision, the Administrator has carefully considered the health effects information contained in the 1993 CD, the 1995 Staff Paper, and the advice and recommendations of the CASAC as presented both in discussion of these documents at public meetings and in its 1995 closure letter (see docket A-93-06).

The EPA staff identified several factors that the Administrator should consider in reaching a decision on whether or not to revise the current primary standard to protect against exposures to NO₂. These factors include: the sensitive populations affected by nitrogen dioxides, the nature and severity of the health effects, and the protection afforded by the current standards.

a. Sensitive Populations Affected.

Two general groups in the population may be more susceptible to the effects of NO₂ exposure than other individuals. These groups include persons with pre-existing respiratory disease and children 5 to 12 years old (SP, 1995, p. 39).

Individuals in these groups appear to be affected by lower levels of NO₂ than individuals in the rest of the population.

Both the 1993 CD and the 1995 Staff Paper support the hypothesis that those with pre-existing respiratory disease have an enhanced susceptibility from exposure to NO₂. Since these individuals live with reduced ventilatory reserves, any reductions in pulmonary function caused by exposure to NO₂ have the potential to further compromise their ventilatory capacity. Compared to healthy individuals with normal ventilatory reserves who may not notice small reductions in lung function, those with pre-existing respiratory disease may be prevented from continuing normal activity following exposure to NO₂.

Asthmatic individuals are considered one of the subpopulations most responsive to NO₂ exposure (CD, 1993, p. 16-1). The National Institutes of Health (1991) estimates that approximately 10 million asthmatics live in the U.S. Because asthmatics tend to be much more sensitive to inhaled bronchoconstrictors than nonasthmatics, there is the added concern that NO₂-induced increase in airway response may exacerbate already existing hyperresponsiveness caused by pre-exposure to other inhaled materials.

Patients with chronic obstructive pulmonary disease (COPD) constitute another subpopulation which is more responsive to NO₂ exposure than the average population. This group, which is estimated to be 14 million in the U.S. (U.S. Department of Health and Human Services, 1990), includes persons with emphysema and chronic bronchitis. One of the major concerns for COPD patients is that they do not have an adequate ventilatory reserve and, therefore, would tend to be more affected by any additional loss of ventilatory function as may result from exposure to NO₂. The available data also indicate that NO₂ might further damage already impaired host defense mechanisms, thus putting COPD patients at increased risk for lung infection.

Numerous epidemiological studies conducted in homes with gas stoves provide evidence that children (5-12 years old) are at increased risk of respiratory symptoms/illness from exposure to elevated NO₂ levels (Melia *et al.*, 1977, 1979, 1983; Ekwo *et al.*, 1983; Ware *et al.*, 1984; Ogston *et al.*, 1985; Dockery *et al.*, 1989a; Neas *et al.*, 1990, 1991, 1992; Dijkstra *et al.*, 1990; Brunekreef *et al.*, 1989; Samet *et al.*, 1993). Because childhood respiratory illness is very common (Samet *et al.*, 1983; Samet and Utell, 1990), any impact which NO₂ might have in

increasing the probability of respiratory illness in children is a matter of public health concern. This is particularly true in light of evidence that recurrent childhood respiratory disease may be a risk factor for later susceptibility to lung damage (Glezen, 1989; Samet *et al.*, 1983; Gold *et al.*, 1989). In the U.S., there are approximately 35 million children in the age group 5 to 14 years (Centers for Disease Control, 1990).

b. *Health Effects of Concern.* Based on the health effects information contained in the 1993 CD (which evaluates key studies published through early 1993) and the 1995 Staff Paper, EPA has concluded that NO₂ is the only nitrogen oxide sufficiently widespread and commonly found in ambient air at high enough concentrations to be a matter of public health concern. Exposure to NO₂ is associated with a variety of acute and chronic health effects. The health effects of most concern at ambient or near-ambient concentrations of NO₂ include changes in airway responsiveness and pulmonary function in individuals with pre-existing respiratory illnesses and increases in respiratory illnesses in children (5–12 years old).

The changes in airway responsiveness and pulmonary function are mostly associated with short-term exposures (e.g., less than 3 hours). Investigations of long-term exposures of animals to NO₂ levels higher than those found in the ambient air provide evidence for possible underlying mechanisms of NO₂-induced respiratory illness such as those observed in the indoor epidemiological studies described below. Furthermore, animal studies have also provided evidence of emphysema caused by long-term exposures to greater than 8 ppm NO₂. The key evidence regarding these effects is summarized below.

(1) Increase in airway responsiveness. There is little, if any, convincing evidence that healthy individuals experience increases in airway responsiveness when exposed to NO₂ levels below 1.0 ppm. However, studies of asthmatics have reported some evidence of increased airway responsiveness caused by short-term exposures (e.g., less than 3 hours) to NO₂ at relatively low concentrations (mostly within the range of 0.2 to 0.3 ppm NO₂) which are of concern in the ambient environment.

Responsiveness of an individual's airways is typically measured by evaluating changes in airway resistance or spirometry following challenge with a pharmacologically-active chemical (e.g., histamine, methacholine, carbachol), which causes constriction of the airways. Airway

hyperresponsiveness is reflected by an abnormal degree of airway narrowing caused primarily by airway smooth muscle shortening in response to nonspecific stimuli. Asthmatics experience airway hyperresponsiveness to certain chemical and physical stimuli and have been identified as one of the population subgroups which is most sensitive to short-term NO₂ exposure (CD, 1993, p. 16–1).

Several controlled human exposure studies (Ahmed *et al.*, 1983a,b; Bylin *et al.*, 1985; Hazucha *et al.*, 1982, 1983; Koenig *et al.*, 1985; Orehek *et al.*, 1981) of asthmatic individuals showed no significant effect on responsiveness at very low NO₂ concentrations of 0.1 to 0.12 ppm. Folinsbee (1992) analyzed data on asthmatics experimentally-exposed to NO₂ in various studies which used challenges producing increased airway responsiveness in 96 subjects and decreased airway responsiveness in 73 subjects. For exposures in the range of 0.2 to 0.3 ppm NO₂, he found that the excess increase in airway responsiveness was attributable to subjects exposed to NO₂ at rest. Because NO₂ at these levels does not appear to cause airway inflammation and the increased airway responsiveness appears fully reversible, implications of the observed increases in responsiveness remain unclear. It has been hypothesized that increased nonspecific airway responsiveness caused by NO₂ could lead to increased responses to a specific antigen; however, there is no plausible evidence to support this.

(2) Decrease in pulmonary function. Nitrogen dioxide induced pulmonary function changes in asthmatic individuals have been reported at low, but not high, NO₂ concentrations. For the most part, the small changes in pulmonary function that have been observed in asthmatic individuals have occurred at concentrations between 0.2 and 0.5 ppm, but not at much higher concentrations (i.e., up to 4 ppm) (CD, 1993, p. 16–3). In one early study of asthmatics, symptoms of respiratory discomfort were experienced by 4 of 13 asthmatics exposed to 0.5 ppm for 2 hours; however, Kerr *et al.* (1979) concluded that the symptoms were minimal and did not correlate well with functional changes. In several other studies of asthmatics, very small changes in spirometry or plethysmography were reported following acute exposures in the range of 0.1 (Hazucha *et al.*, 1982, 1983) to 0.6 ppm NO₂ (Avol *et al.*, 1988). Hazucha found an 8 percent increase in specific airway resistance (SR_{aw}) after mild asthmatics were exposed to 0.1 ppm

NO₂ at rest. However, this finding is not considered statistically significant. Bauer *et al.*, (1986) reported statistically significant changes in spirometric response in mild asthmatics exposed for 20 minutes (with mouthpiece) to 0.3 ppm NO₂ and cold air. Avol *et al.* (1988) found significant changes in SR_{aw} and 1-second forced expiratory volume (FEV₁) as a function of exposure concentration and duration for all exposure conditions (i.e., exposure of moderately exercising asthmatics for 2 hours to 0.3 ppm and 0.6 ppm NO₂); however, it was concluded that there was no significant effect of NO₂ exposure on these measures of pulmonary function (CD, 1993, p. 15–47). Exercising adolescent asthmatics exposed (with mouthpiece) to air, 0.12 ppm and 0.18 ppm NO₂, exhibited small changes in FEV₁, but there were no differences in symptoms between air and either of the NO₂ exposures (Koenig *et al.*, 1987a,b). The absence of spirometry or plethysmography changes in studies (Avol *et al.*, 1986; Bylin *et al.*, 1985; Linn *et al.*, 1985b; Linn *et al.*, 1986) conducted at higher NO₂ concentrations makes developing a concentration-response relationship problematic (CD, 1993, p. 15–62). In assessing the available data on pulmonary function responses to NO₂ in asthmatic individuals, the CD concludes that the most significant responses to NO₂ that have been observed in asthmatics have occurred at concentrations between 0.2 and 0.5 ppm (CD, 1993, p. 16–3).

Patients with COPD experience pulmonary function changes with brief exposure to high concentrations (5 to 8 ppm for 5 minutes) or with more prolonged exposure to lower concentrations (0.3 ppm for 3.75 hours).

(3) Increased occurrence of respiratory illness among children. Epidemiological evidence includes a meta-analysis of nine epidemiological studies of children (5–12 years old) living in homes with gas stoves. The meta-analysis reported that children (ages 5–12 years) living in homes with gas stoves have an increased risk of about 20 percent for developing respiratory symptoms and disease over children living in homes without gas stoves. This increase in risk corresponds to each increase of 0.015 ppm NO₂ in estimated 2-week average NO₂ exposure, where mean weekly concentrations in bedrooms reporting NO₂ levels were predominantly between 0.008 and 0.065 ppm NO₂ (CD, 1993, p. 14–73). A detailed discussion of the studies included in the meta-analysis can be found in the 1993 CD as well as in the 1995 Staff Paper.

In assessing the potential value of the meta-analysis in developing the basis for a NAAQS for NO₂, the Administrator is mindful of the limitations of the underlying studies. As discussed in the CD and Staff Paper, the gas stove studies do not provide sufficient exposure information, including human activity patterns, to establish whether the observed health effects are related primarily to peak, repeated peak, or lower, long-term, average exposures to NO₂. Furthermore, both the staff and CASAC concurred that, absent information on exposure patterns in the gas stove studies, it is not reasonable to extrapolate the results of these indoor studies to outdoor exposure regimes (SP, 1995). Indoor exposure patterns to NO₂ are quite different compared to outdoor exposure patterns. With potentially much higher peaks and average indoor exposures than would be found outdoors, it is extremely difficult to extrapolate the results of the meta-analysis in a manner which would provide quantitative estimates of health impacts for outdoor exposures to NO₂ (CD, 1993, p. 16-5).

(4) Biological Plausibility. Animal toxicology studies provide evidence for possible underlying mechanisms of NO₂-induced respiratory illness. These studies have shown that exposure to NO₂ can impair components of the respiratory host defense system and increase susceptibility to respiratory infection. The increased respiratory symptoms and illness in children reported in the epidemiology studies cited above may be a reflection of the increased susceptibility to respiratory infection caused by the impact of NO₂ on pulmonary defenses. Studies that provide a plausible biological basis for developing such a hypothesis and that highlight the potential effects associated with long-term exposures to NO₂ are discussed in detail in the 1993 CD and 1995 Staff Paper.

Although the pulmonary immune system has not been adequately studied to assess the impact of NO₂ exposure, there is some indication that NO₂ suppresses some systemic immune responses and that these responses may be both concentration and time dependent. In the ambient range of exposures, time may be a more important influence than concentration. However, there were no data showing clearly the effect of time on effects of long-term, low-level exposures representing ambient exposure levels.

In the urban air, the typical pattern of NO₂ is a low-level baseline exposure on which peaks are superimposed. When the relationship of the peak to baseline exposure and of enhanced susceptibility

to bacterial infection was investigated, the results indicated that no simplistic concentration times time relationship was present, and that peaks had a major influence on the outcome (Gardner, 1980; Gardner *et al.*, 1982; Graham *et al.*, 1987). Several other animal infectivity studies (Miller *et al.* 1987; Gardner *et al.*, 1982; Graham *et al.*, 1987) offered evidence which indicated that mice exposed to baseline plus short-term peaks were more susceptible to respiratory infection than either those exposed to control or background levels of NO₂. This research also indicated that the pattern of NO₂ exposure had a major influence on the response.

The weight of evidence provided by animal toxicology supports the contention that NO₂ impairs the ability of host defense mechanisms to protect against respiratory infection. Although some of the health endpoints may not be valid for humans (e.g., increased mortality), there are many shared mechanisms between animals and humans which support the hypothesis of association between NO₂ exposure and increases in respiratory symptoms and illness reported in the epidemiological studies.

Based on the information reviewed in the CD and the Staff Paper, it is clear that at sufficiently high concentrations of NO₂ (i.e., > 8 ppm) for long periods of exposure, NO₂ can cause morphologic lung lesions in animals that meet the criteria for a human model of emphysema (which requires the presence of alveolar wall destruction in addition to enlargement of the airspace distal to the terminal bronchiole). Although current information does not permit identification of the lowest NO₂ levels and exposure periods which might cause emphysema, it is apparent that levels required to induce emphysematous lung lesions in animals are far higher than any NO₂ levels which have been measured in the ambient air.

c. *Air Quality Considerations.* One of the factors the Administrator considered in reaching this proposed decision is the relationship between short-term exceedances of NO₂ concentrations and the annual NO₂ mean. In 1994, McCurdy analyzed air quality data from the period 1988-1992 to determine the estimated number of exceedances of various NO₂ short-term air quality indicators which would occur given attainment of a range of annual averages. The annual averages McCurdy analyzed ranged from 0.02 to 0.06 ppm and included the current NO₂ NAAQS of 0.053 ppm. The 1-hour and daily concentration levels chosen for analyses were 0.15, 0.20, 0.25, and 0.30 ppm. The

results of this analysis are reported in "Analysis of High 1 Hr NO₂ Values and Associated Annual Averages Using 1988-1992 Data" (McCurdy, 1994). In his report, McCurdy concluded that areas attaining the current annual NO₂ NAAQS reported few, if any, 1 hour or daily exceedances above 0.15 ppm.

Los Angeles is the only city in the U.S. to record violations of the annual average NO₂ NAAQS during the past decade. However, in 1992, Los Angeles reported air quality measurements which meet the NO₂ NAAQS for the first time. Thus, currently, the entire U.S. is in attainment of the current NO₂ NAAQS.

d. *Proposed Decision on the Primary Standard.* Based on the assessment of the health and air quality information presented in the CD and Staff Paper and discussed above, and taking into account the advice and recommendations of EPA staff and CASAC, the Administrator has determined pursuant to section 109(d)(1) of the Act, as amended, that it is not appropriate to propose any revision of the existing annual primary standard for NO₂ at this time.

In reaching this proposed decision, the Administrator took into account that the existing standard level is well below those levels associated with chronic effects observed in animal studies. The current standard also provides substantial protection against those short-term peak NO₂ concentrations at which clinical studies found statistically-significant changes in pulmonary function or airway responsiveness. As part of the review of the primary standard, the Administrator also considered whether a new short-term standard for NO₂ would be appropriate. Based on the available air quality data, the Administrator concluded that the existing annual standard provides adequate protection against potential changes in pulmonary function or airway responsiveness (which most experts would characterize as mild responses occurring in the range of 0.2 to 0.5 ppm NO₂). The adequacy of the existing annual standard to protect against potential pulmonary effects is further supported by the absence of documented effects in some studies at higher (3 to 4 ppm NO₂) concentrations (SP, 1995, p. 43).

In reviewing the scientific bases for an annual standard, the Administrator finds that the evidence showing the most serious health effects associated with long-term exposures (e.g., emphysematous-like alterations in the lung and increased susceptibility to infection) comes from animal studies conducted at concentrations well above

those permitted in the ambient air by the current standard. While recognizing there is no satisfactory method for quantitatively extrapolating exposure-response results from these animal studies directly to humans, the Administrator is concerned that there is some risk to human health from long-term exposure to elevated NO₂ levels given the potential seriousness of the effects in animals.

Other evidence suggesting health effects related to long-term, low-level exposures, such as the epidemiological studies integrated into the meta-analysis, provides some qualitative support for concluding that there is a relationship between long-term human exposure to near-ambient levels of NO₂ and adverse health effects. However, the various limitations in these studies preclude derivation of quantitative dose-response relationships for the ambient environment. The Administrator is mindful that there remains substantial uncertainty about the actual exposures of subjects in the studies that make up the meta-analysis. The NO₂ levels which were monitored in the gas-stove studies are only estimates of exposure and do not represent actual exposures. Because the studies collected 2-week average NO₂ measurements, one cannot distinguish between relative contributions to respiratory symptoms and illness of peak, repeated peak and long-term average exposure to NO₂. In addition, indoor exposure patterns to NO₂ are quite different compared to outdoor exposure patterns. With potentially much higher peaks and average indoor exposures than would be found outdoors, it is extremely difficult to extrapolate the results of the meta-analysis in a manner which would provide quantitative estimates of health impacts for outdoor exposures to NO₂ (CD, 1993, p. 16–5). Given these limitations, the Administrator concurs with the EPA staff and CASAC that neither the meta-analysis nor the underlying studies provide a quantitative basis for standard setting purposes. In her judgement, they do, however, provide qualitative support for the retention of the existing standard which provides protection against both peaks and long-term NO₂ exposures.

In reaching this proposed decision, the Administrator also took into account that the available air quality data indicate that if the existing standard of 0.053 ppm NO₂ is attained, the occurrence of 1-hour NO₂ values greater than 0.2 ppm would be unlikely in most areas of the country (McCurdy, 1994). The Administrator also considered that

all areas of the U.S. are in attainment of the current NO₂ NAAQS.

After carefully assessing the available health effects and air quality information, it is the Administrator's judgment that a 0.053 ppm annual standard would keep annual NO₂ concentrations considerably below the long-term levels for which serious chronic effects have been observed in animals. Retaining the existing standard would also provide protection against short-term peak NO₂ concentrations at the levels associated with mild changes in pulmonary function and airway responsiveness observed in controlled human studies. In reaching this judgment, the Administrator fully considered the 1995 Staff Paper conclusions with respect to the primary standard and the views of the CASAC (Wolff, 1995). For the above reasons, the Administrator has determined, under section 109(d)(1) of the Act, as amended, that it is not appropriate to propose any revision of the existing primary standard for NO₂ of 0.053 ppm annual average at this time.

B. The Secondary Standard

Nitrogen dioxide and other nitrogen compounds have been associated with a wide range of effects on public welfare. The effects associated with nitrogen deposition include acidification and eutrophication of aquatic systems, potential changes in the composition and competition of some species of vegetation in wetland and terrestrial systems, and visibility impairment. The direct effects of NO₂ on vegetation and materials are also considered. The CD and Staff Paper discuss in detail the major effects categories of concern; the following discussion draws from these documents.

1. Direct Effects of Nitrogen Dioxides

a. *Vegetation.* Data evaluated in the 1993 CD indicate that single exposures to NO₂ for less than 24 hours can produce effects on the growth, development, or reproduction of plants at concentrations that greatly exceed the ambient levels of NO₂ observed in the U.S. In experiments of 2 weeks or more, with intermittent exposures of several hours per day, effects on growth or yield start to appear when the concentration of NO₂ reaches the range of 0.1 to 0.5 ppm, depending on the species of plant and conditions of exposure (CD, 1993, p. 9–89).

As reported in the 1993 CD (pp. 9–113 to 9–137), several studies have examined synergistic or additive effects of NO₂ and other air pollutants on plants. These studies report that NO₂ in combination with other pollutants (i.e.,

sulfur dioxide, ozone) can increase plant sensitivity, thus lowering concentration and time of exposure required to produce injury/growth effects. The pollutant concentrations used in these experimental studies were well above those observed in the ambient air and at frequency of co-occurrence that are not typically found in the U.S. (CD, 1993, p. 9–127).

b. *Materials.* Nitrogen oxides are known to enhance the fading of dyes; diminish the strength of fabrics, plastics, and rubber products; assist the corrosion of metals; and reduce the use-life of electronic components, paints, and masonry. Compared to studies on sulfur oxides, however, there is only limited information available quantifying the effects of nitrogen oxides. While NO₂ has been qualitatively associated with materials damage, it is difficult to distinguish a single causative agent for observed damage to exposed materials because many agents, together with a number of environmental stresses, act on a surface throughout its life.

c. *Conclusions Concerning Direct Effects on Vegetation and Materials.* Based on the information assessed in the CD and Staff Paper and taking into account the advice and recommendations of EPA staff and CASAC, the Administrator has determined that the existing annual secondary standard appears to be both adequate and necessary to protect against the direct effects of NO₂ on vegetation and materials, and that it is not appropriate to propose any modifications of the secondary standard with respect to such effects. In reaching this proposed decision, the Administrator considered evidence indicating that attainment of the existing annual secondary standard provides substantial protection against both long-term and peak NO₂ concentrations which may lead to the direct effects described above.

d. *Other Related Effects of Nitrogen Dioxide.* While NO₂ can contribute to brown haze, the available scientific evidence indicates that light scattering by particles is generally the primary cause of degraded visual air quality and that aerosol optical effects alone can impart a reddish-brown color to a haze layer. Because of this, the improvement in visual air quality to be gained by reducing NO₂ concentrations is highly uncertain at best. In addition, as discussed in the 1995 Staff Paper, there is no established relationship between ground level NO₂ concentrations at a given point and visibility impairment due to a plume or regional haze. These considerations led both the EPA staff

and CASAC to conclude that establishment of a secondary NO₂ standard to protect visibility would not be appropriate. The Administrator concurs with those judgments.

While concluding that a secondary NO₂ standard is not appropriate to protect visibility, the Administrator is concerned about visibility impairment in our national parks and wilderness areas. To address visible plumes that impact the visual quality of Class I areas, EPA adopted regulations (under section 165(d) of the Act) in 1980. In addition, EPA is in the process of developing regional haze regulations under section 169A of the Act.

2. Nitrogen Deposition

As summarized below, the deposition of nitrogen compounds contributes to a wide range of environmental problems. As discussed in detail in the 1993 CD and 1995 Staff Paper, nitrogen compounds effect terrestrial, wetland, and aquatic ecosystems through direct deposition or by indirectly altering the complex biogeochemical nitrogen cycle. In assessing the available effects information evaluated in the CD and Staff Paper, the Administrator is mindful of the scientific complexity of nitrogen deposition issues and their broad implications for the environment.

Nitrogen moves through the biosphere via a complex series of biologically and non-biologically mediated transformations. The processes that make up the nitrogen cycle and transform nitrogen as it moves through an ecosystem include: assimilation, nitrification, denitrification, nitrogen fixation, and mineralization. Similar types of transformations can be found in diverse habitats, but the organisms responsible for the transformations and the rates of the transformations themselves can vary greatly.

Atmospheric deposition of nitrogen can disturb the nitrogen cycle and result in the acidification of soils, lakes, and streams. It can also lead to the eutrophication of sensitive estuarine ecosystems by changing vegetation composition and affecting nutrient balance. Because a great degree of diversity exists among ecosystem types, as well as in the mechanisms by which these systems assimilate nitrogen inputs, the time to nitrogen saturation (i.e., nitrogen input in excess of total combined plant and microbial nutritional demands) will vary from one system or site to another. As a consequence, the relationship between nitrogen deposition rates and their potential environmental impact is to a large degree site or regionally-specific and may vary considerably over broader

geographical areas or from one system to another because of the amount, form, and timing of nitrogen deposition, forest type and status, soil types and status, the character of the receiving waterbodies, the history of land management and disturbances across the watersheds and regions, and exposure to other pollutants. Absent better quantification of these factors, it is difficult to link specific nitrogen deposition rates with observed environmental effects, particularly at the national level.

a. *Terrestrial/Wetland.* The principal effects on soils and vegetation associated with excess nitrogen inputs include: (1) Soil acidification and mobilization of aluminum, (2) increase in plant susceptibility to natural stresses, and (3) modification of inter-plant competition. Atmospheric deposition of nitrogen can accelerate the acidification of soils and increase aluminum mobilization if the total supply of nitrogen to the system (including deposition and internal supply) exceeds plant and microbial demand. However, the levels of nitrogen input necessary to produce measurable soil acidification are quite high. As reported in the Criteria Document (Tamm and Popovic, 1974; Van Miegroet and Cole, 1984), it is estimated that nitrogen inputs ranging from 50 to 3,900 kilograms per hectare (kg/ha) for 50 and 10 years respectively, would be required to affect a change in soil potential for hydrogen (pH) of 0.5 pH units. At present, nitrogen deposition has not been directly associated with the acidification of soils in the U.S. The potential exists, however, if additions are high enough for sufficiently long periods of time, particularly in areas where soils have low buffering capacity. Mobilization of aluminum can be toxic to plants and, if transported to waterways, can be toxic to various aquatic species (SP, 1995, pp. 64,65).

Several studies evaluated in the CD and Staff Paper examined the effects of nitrogen deposition on forest species sensitivity to drought, cold, or insect attack. While some studies (Margolis and Waring, 1986; De Temmerman et al., 1988; Waring and Pitman, 1985; White, 1984) report that increased nitrogen deposition can alter tree susceptibility to frost damage, insect and disease attack, and plant community structure, other studies (Klein and Perkins, 1987; Van Dijk et al., 1990) did not. For example, Margolis and Waring showed that fertilization of Douglas fir with nitrogen could lengthen the growing season to the point where frost damage became a problem. However, Klein and Perkins presented

other evidence that showed no additional winter injury of high elevation conifer forests when fertilized with 40 kilogram total nitrogen/ha/year. On the other hand, De Temmerman et al. provided data showing increased fungal outbreaks and frost damage on several pine species exposed to very high ammonia deposition rates (> 350 kg/ha/year). Numbers of species and fruiting bodies of fungi have also increased concomitantly with nitrogen deposition in Dutch forests (Van Breeman and Van Dijk, 1988). The CD evaluated a number of other studies which also gave mixed results as to the impact of excessive inputs of nitrogen into forest ecosystems (CD, 1993, pp. 10-92,93).

Climate is thought to play a major role in the severe red spruce decline in the Northeastern U.S., perhaps with some additional exacerbation due to the direct effects of acid mist on foliage (Johnson et al., 1992). There is also some evidence that suggests that indirect effects of nitrogen saturation, namely nitrate and aluminum leaching, may be contributing factors to red spruce decline in the Southern Appalachians (CD, 1993, p. 10-74).

In wetland ecosystems, primary biomass production is most commonly limited by the availability of nitrogen. Several fertilization studies have reported that nitrogen application can result in changes in species composition or dominance in wetland systems. Vermeer (1986) found that in fen and wet grassland communities, grasses tended to increase in dominance over other species. Jefferies and Perkins (1977) also found a species-specific change in stem density at a Norfolk, England, salt marsh after fertilizing monthly with 610 kg NO₃ nitrogen/ha/year or 680 kg NH₄+ nitrogen/ha/year over a period of 3 to 4 years.

Long-term studies (greater than 3 years) of increased nitrogen loadings to wetland systems have reported that increases in primary production can result in changes in species composition and succession (U.S. EPA, 1993, pp. 10-120-121). Changes in species composition may occur from increased evapotranspiration (Howes et al., 1986; Logofet and Alexander, 1984) leading to a changed water regime that favors different species or from increased nutrient loss from the system through incorporation into or leaching from aboveground vegetation. In parts of Europe, historical data seem to implicate pollutant nitrogen in altering the competitive relationships among plants and threatening wetland species adapted to habitats of low fertility

(Tallis, 1964; Ferguson et al., 1984; Lee et al., 1986).

Potential changes in species composition and succession in wetlands is of particular concern because wetlands are habitats to many rare and threatened plant species. Some of these plants have adapted to systems low in nitrogen or with low nutrient levels. For some species, these conditions can be normal for growth. Therefore, excess nitrogen deposition can alter these conditions and thus alter species density and diversity. In the contiguous U.S., wetlands harbor 14 percent (18 species) of the total number of plant species that are formally listed as endangered. Several species on this list, such as the insectivorous plants, are widely recognized to be adapted to nitrogen-poor environments. While changes in species composition and succession are of concern, such changes have not been associated with nitrogen deposition in the U.S.

b. *Aquatic*. Some aquatic systems are potentially at risk from atmospheric nitrogen additions through the processes of eutrophication and acidification. Both processes can sufficiently reduce water quality making it unfit as a habitat for most aquatic organisms and/or human consumption. Acidification of lakes from nitrogen deposition may also increase leaching and methylation of mercury in aquatic systems.

Atmospheric nitrogen can enter aquatic systems either as direct deposition to water surfaces or as nitrogen deposition to the watershed. In northern climates, nitrate may be temporarily stored in snow packs and released in a more concentrated form during snow melt. Nitrogen deposited to the watershed is then routed (e.g., through plant biomass and soil microorganisms) and transformed (e.g., into other inorganic or organic nitrogen species) by watershed processes, and may eventually run off into aquatic systems in forms that are only indirectly related to the original deposition. The contributions of direct and indirect atmospheric loadings have received increased attention. While the available evidence indicates that the impact of nitrogen deposition on sensitive aquatic systems can be significant, it is difficult to quantify the relationship between atmospheric deposition of nitrogen, its appearance in receiving waters, and observed effects.

(1) *Acidification*. In the U.S., the most comprehensive assessment of chronic acidification of lakes and streams comes from the National Surface Water Survey (NSWS) conducted as part of the National Acid Precipitation Assessment

Program (NAPAP). A detailed discussion of the findings in the NSWS can be found in both the 1993 CD and the 1995 Staff Paper. The studies highlighted in these documents reported mixed observations as to the relative contribution of nitrogen compounds to chronic acidification in North American lakes. However, the National Stream Survey (NSS) data do suggest that the Catskills, Northern Appalachians, Valley and Ridge Province, and Southern Appalachians all show some potential for chronic acidification due to nitrate ions (NO_3). Two studies (Kaufmann et al., 1991; Driscoll et al., 1989) have examined whether atmospheric deposition is the source of the NO_3 leaking out of these watersheds. Data from the NSS (Kaufmann et al., 1991) suggest a strong correlation between concentrations of stream water and levels of wet nitrogen deposition in each of the NSS regions. Secondly, Driscoll et al. (1989) collected input/output budget data for a large number of watersheds in the U.S. and Canada and summarized the relationship between nitrogen export and nitrogen deposition at all the sites. Though the relationships discovered should not be over-interpreted or construed as an illustration of cause and effect, they do show that watersheds in many regions of North America are retaining less than 75 percent of the nitrogen that enters them, and that the amount of nitrogen being leaked from these watersheds is higher in areas where nitrogen deposition is highest.

On a chronic basis in the U.S., especially in the eastern part of the country, nitrogen deposition does play a role in surface water acidification. However, there are significant uncertainties with regard to the long-term role of nitrogen deposition in surface water acidity and with regard to the quantification of the magnitude and timing of the relationship between atmospheric deposition and the appearance of nitrogen in surface waters.

Episodic acidification in surface waters is a concern in the Northeast, Mid-Atlantic, Mid-Atlantic Coastal Plain, Southeast, Upper Midwest, and West regions (Wigington et al., 1990). In the Mid-Atlantic Coastal Plain and Southeast regions, all of the episodes reported to date have been associated with rainfall. In contrast, most of the episodes in the other regions are related to snowmelt, although rain-driven episodes apparently can occur in all regions of the country. It is important to stress that even within a given area, such as the Northeast, major differences can be evident in the occurrence,

nature, location (lakes or streams), and timing of episodes at different sites. The 1995 Staff Paper provides a detailed description of the processes which may contribute to the timing and severity of acidic episodes.

Some broad geographic patterns in the frequency of episodes in the U.S. are now evident. Episodes driven by NO_3 are common in the Adirondacks and Catskill Mountains of New York, especially during snowmelt, and also occur in at least some streams in other portions of the Northeast (e.g., Hubbard Brook). Nitrate contributes on a smaller scale to episodes in Ontario and may play some role in episodic acidification in the Western U.S. There is little current evidence that NO_3 episodes are important in the acid-sensitive portions of the Southeastern U.S. outside the Great Smoky Mountains. There is no information on the relative contribution of NO_3 to episodes in many of the subregions covered by the NSS, including those that exhibited elevated NO_3 concentrations at spring base flow (e.g., the Appalachian Plateau, the Valley and Ridge Province and Mid-Atlantic Coastal Plain), because temporally-intensive studies have not been published for these areas.

While the available data suggest that NO_3 episodes are more severe now than they were in the past, it is important to emphasize that only the data reported for the Catskills can be considered truly long-term (up to 65 years of record). Data for the Adirondacks (Driscoll and Van Dreason, 1993) and other areas of the U.S. (Smith et al., 1987) span only 1 to 2 decades and should be interpreted with caution.

Because surface water nitrogen increases have occurred at a time when nitrogen deposition has been relatively unchanged in the Northeastern U.S. (Husar, 1986; Simpson and Olsen, 1990), it is suggestive that nitrogen saturation of watersheds is progressing and that current levels of nitrogen deposition are too high for the long-term stability of aquatic systems in the Adirondacks, the Catskills, and possibly elsewhere in the Northeast. It is important to note that this supposition is dependent on our acceptance of NO_3 episodes as evidence of nitrogen saturation. While there is some support for this, there are significant uncertainties with respect to the quantification of the linkage and the timing of the relationship between the atmospheric deposition of nitrogen and its episodic or chronic appearance in surface waters.

This relationship between deposition and effect becomes more complex because the capacity to retain nitrogen

differs from one watershed to another and from one region to another as watershed and regional features differ. The differing features that may contribute to these differences include, the amount, form and timing of nitrogen deposition, forest type and status (including soil type and status), the character of the receiving waterbodies, the history of land management and disturbances across watersheds and regions and exposure to other pollutants. For example, the Northeast, because of the presence of aggrading forests and deeper soils in comparison to those of the West, may be able to absorb higher rates of deposition without serious effects than areas of the mountainous West, where soils are thin in comparison and forests are often absent at the highest elevations (CD, p. 10–179). The data of Silsbee and Larson (1982) suggest strongly that forest maturation is also linked to the process of NO₃ leakage from Great Smoky Mountain watersheds.

In summary, the available data indicate that nitrogen contributes to episodic acidification of sensitive streams and lakes in the Northeast. The data also suggest that some watersheds of the Northeast and the mid-Appalachians may be nearing nitrogen saturation. If, and when, this occurs, nitrogen deposition will become a more direct cause of chronic surface water acidification. At present, however, it is difficult to establish quantitative relationships between nitrogen deposition and the appearance of nitrogen in receiving waters, given the uncertainties in determining time to nitrogen saturation for varying systems and sites. The complexity of the scientific issues involved led the CASAC to conclude that available scientific information assessed in the Criteria Document and Staff Paper did not provide an adequate basis for standard setting purposes at this time (see Wolff, 1995). In its review of the Acid Deposition Standard Feasibility Study: Report to Congress (U.S. EPA, 1995), the Acid Deposition Effects Subcommittee of the Ecological Processes and Effects Committee of the EPA's Science Advisory Board also concluded that there was not an adequate scientific basis for establishing an acidic deposition standard (see "An SAB Report: Review of the Acid Deposition Standard Feasibility Study Report to Congress," U.S. EPA, 1995).

(2) *Eutrophication*. Eutrophication is the process by which aquatic systems are enriched with the nutrient(s) that are presently limiting for primary production in that system. Eutrophication may produce conditions

of increased algal biomass and productivity, nuisance algal populations, and decreases in oxygen availability for heterotrophic organisms. Another effect of chronic eutrophication is increased algal biomass shading out ecologically-valuable estuarine seagrass beds. Eutrophy can lead to fish kills and the permanent loss of some sensitive species caused by suffocation or rarely because of some kind of toxic algal bloom. Though this process often occurs naturally over the long-term evolution of lakes, it can be significantly accelerated by the additional input of the limiting nutrients from anthropogenic sources. In order to establish a link between nitrogen deposition and the eutrophication of aquatic systems, one must first demonstrate that the increase in biomass within the system is limited by nitrogen availability, and second, that nitrogen deposition is a major source of nitrogen to the system.

In most freshwater systems, phosphorus, not nitrogen, is the limiting nutrient. Therefore, eutrophication by nitrogen inputs will only be a concern in lakes that are chronically nitrogen limited and have a substantial total phosphorous concentration. This condition is common only in lakes that have received excessive inputs of anthropogenic phosphorous, or in rare cases, have high concentrations of natural phosphorous. In the former case, the primary dysfunction of the lakes is an excess supply of phosphorous, and controlling nitrogen deposition would be an ineffective method of gaining water quality improvement. In the latter case, lakes with substantial total phosphorous concentrations would experience measurable increases in biomass from increases in nitrogen deposition.

In contrast to freshwater systems, the productivity of estuarine waters of the U.S. correlates more closely with supply rates of nitrogen than of other nutrients (Nixon and Pilson, 1983). Because estuaries and coastal waters receive substantial amounts of weathered material from terrestrial ecosystems and from exchange with sea water, acidification is not a concern. However, this same load of weathered material and anthropogenic inputs makes these same areas prone to the effects of eutrophication.

Considerable research has focused on whether estuarine and coastal ecosystems are limited by nitrogen, phosphorus, or some other factor. Numerous geochemical and experimental studies have suggested that nitrogen limitation is much more common in estuarine and coastal waters

than in freshwater systems (CD, 1993, pp. 10–189 to 197). However, specific instances of phosphorus limitation (Smith, 1984) and of seasonal switching between nitrogen and phosphorus limitation (D'Elia *et al.*, 1986; McComb *et al.*, 1981) have been observed.

Estimation of the contribution of nitrogen deposition to the eutrophication of estuarine and coastal waters is made difficult by the multiple direct anthropogenic sources (e.g., from agriculture and sewage) of nitrogen. In the U.S., only a few systems have been studied with enough intensity to develop predictions about the contribution of atmospheric nitrogen to total nitrogen inputs. One example is the Chesapeake Bay, where a large effort has been made to establish the relative importance of different sources of nitrogen to the total nitrogen load entering the bay (e.g., D'Elia *et al.*, 1982; Smullen *et al.*, 1982; Fisher *et al.*, 1988a; Tyler, 1988). The signatories to the Chesapeake Bay Agreement (i.e., Maryland, Virginia, Pennsylvania, the District of Columbia, and EPA, through their Baywide Nutrient Reduction Strategy and individual tributary watershed nutrient reduction strategies) have committed to reduce nitrogen and phosphorus loadings to the bay by 40 percent (from 1985 baseline) by the year 2000.

Enhanced modeling is being used to better assess source responsibility for the transport and deposition of nitrogen from the 350,000 square miles Chesapeake Bay airshed. This enhanced modeling will assist EPA in deciding: (1) Whether to include reductions in atmospheric NO_x and resultant decreased loadings via atmospheric deposition in the reductions of total nitrogen loading necessary to achieve the planned 40 percent reduction goal by the year 2000, and (2) the role implementation of the Act will play in ensuring nitrogen loadings are capped at the 40 percent reduction goal beyond the year 2000 in the face of significant projected population increases within the Chesapeake Bay watershed (and surrounding airshed). This integration of modeling, watershed, and airshed management will serve as a case study and a prototype method for other geographic areas.

Though estimates for each individual source are very uncertain, studies undertaken to determine the proportion of the total NO₃ load to the bay, which was attributable to nitrogen deposition, produced estimates in the range of 18 to 39 percent. These estimates, which reflect the current status of the area, suggest that supplies of nitrogen from deposition exceed supplies from all

other non-point sources (i.e., farm runoff) to the bay and only point-source inputs (i.e., discharges to water, emissions from industrial facilities) represent a greater input than deposition.

Based on the available data, it is clear that atmospheric nitrogen inputs to estuarine and coastal ecosystems are of concern. The importance of atmospheric inputs will vary, however, from site to site and will depend on the availability of other growth nutrients, the flushing rate through the system, the sensitivity of resident species to added nitrogen, the types and chemical forms of nitrogen inputs from other sources, as well as other factors. Given these complexities, site-specific investigations, such as the Chesapeake Bay Study, are needed to ascertain the most effective mitigation strategy. Similar place-based studies are already under way in the Tampa Bay and other coastal areas.

3. Direct Toxic Effects of Ammonia Deposition to Aquatic Systems

Nitrogen deposition could potentially contribute directly to toxic effects in surface waters. High ammonia concentrations are associated with lesions in gill tissue, reduced growth rates of trout fry, reduced fecundity (number of eggs), increased egg mortality, and increased susceptibility of fish to other diseases, as well as a variety of pathological effects in invertebrates and aquatic plants. Given current maximal concentrations of ammonium ions (NH_4^+) in wet deposition and reasonable maximum rates of dry deposition, even if all nitrogen species were ammonified, the maximum potential NH_4^+ concentrations attributable to deposition would be approximately 280 nmol/L and would be unlikely to be toxic except in unusual circumstances. Therefore, it appears that the potential for toxic effects directly attributable to nitrogen deposition in the U.S. is very limited. In addition, EPA has established water quality standards for ammonia to protect against these effects (50 FR 30784, July 29, 1984; also see guidance document EPA-440/5-85-001).

4. Proposed Decision on the Secondary Standard

As discussed above, after carefully considering the information on the direct effects of NO_2 , the Administrator has determined that the existing annual secondary standard is both necessary and adequate to protect vegetation and materials from the direct effects of NO_2 . The Administrator has also determined

that establishment of a secondary NO_2 standard to protect visibility is not appropriate. In reaching these provisional conclusions, the Administrator has assessed the evidence provided in the CD and the Staff Paper as well as the advice and recommendations of the EPA staff and CASAC.

With respect to nitrogen deposition, the Administrator is concerned about the growing body of scientific information, assessed in the CD and Staff Paper and discussed above, that associates nitrogen deposition with a wide range of environmental effects. Of particular concern is the available data that indicate nitrogen deposition plays a significant role in the episodic acidification of certain sensitive streams and lakes and could cause long-term chronic acidification of such surface waters. The Administrator notes, as did CASAC, that because of the variations in the actual rate of nitrogen uptake, immobilization, denitrification, and leaching, it is very difficult, given current quantification of these processes, to link specific nitrogen deposition rates with observed environmental effects.

In considering the available data, the Administrator is also mindful, given the complex processes involved, that the time to nitrogen saturation will vary from one system to another. As a consequence, the relationship between nitrogen deposition rates and their potential environmental impact is to a large degree site- or regionally-specific and may vary considerably over broader geographical areas. These complexities led both the EPA and CASAC to conclude that there is currently insufficient information to set a national secondary NO_2 standard which would protect against the acidification effects of nitrogen deposition. Because of the site- and regional-specific nature of the problem, the staff also questioned whether adoption of a national secondary NO_2 standard would be an effective tool to address such effects.

In considering the staff's latter view, the Administrator also recognizes that Congress reserved judgment regarding the possible need for further action to control acid deposition beyond the provisions of title IV of the 1990 Amendments and what form any such action might take (Pub. L. 101-549, sec. 404, 104 Stat. 2399, 2632 (1990)). For a more complete discussion of the congressional deliberation on the acidic deposition issue, see 58 FR 21356-21357, April 21, 1993. Among other things, Congress directed EPA to conduct a study of the feasibility and effectiveness of an acid deposition

standard(s), to report to Congress on the role that a deposition standard(s) might play in supplementing the acidic deposition program adopted in title IV, and to determine what measures would be needed to integrate it with that program. The resulting document entitled, "Acid Deposition Standard Feasibility Study: Report to Congress" (U.S. EPA, 1995), concluded, as did the CD and staff paper, that nitrogen deposition plays a significant role in the acidification of certain sensitive streams and lakes and that the time to nitrogen saturation varies significantly from one system or region to another. The complexities of watershed nitrogen dynamics (e.g., the biological processes) and the uncertainties in modeling results that project future effects of nitrogen deposition under alternative emission scenarios, however, led EPA staff (as well as the Acid Deposition Effects Subcommittee of the Ecological Processes and Effects Committee of the EPA's Science Advisory Board that reviewed the report) to conclude that current scientific uncertainties associated with determining the level(s) of an acid deposition standard(s) are significant (see "An SAB Report: Review of the Acid Deposition Standard Feasibility Study Report to Congress," U.S. EPA, 1995). The study does not advocate setting an acid deposition standard at this time. The study does, however, set forth a range of regionally-specific goals to help guide the policy maker when assessing NO_x control strategies and their potential for reducing nitrogen deposition effects.

The Administrator has also examined the available information that indicates atmospheric nitrogen deposition can play a significant role in the eutrophication of estuarine and coastal waters. However, estimation of the contribution of nitrogen deposition to the eutrophication of estuarine and coastal waters is made difficult by multiple direct anthropogenic sources of nitrogen. Thus, the importance of atmospheric inputs will vary from site to site and will depend on the availability of other growth nutrients, the flushing rate through the system, the sensitivity of resident plant species to added nitrogen, as well as the types of chemical forms of nitrogen inputs from other sources. Given the complexities of these factors and the limited data currently available, the Administrator concurs with the EPA staff and CASAC conclusion that there is not sufficient quantitative information to establish a national secondary standard to protect sensitive ecosystems from the eutrophication effects caused by

nitrogen deposition. Rather, additional site-specific investigations (such as the Chesapeake Bay Study) are needed to ascertain the most effective mitigation strategies.

For the above reasons, the Administrator has determined pursuant to section 109(d)(1) of the Act, as amended, that it is not appropriate to propose any revision of the current secondary standard for NO₂ to protect against welfare effects at this time. As provided for under the Act, the EPA will continue to assess the scientific information on nitrogen-related effects as it emerges from ongoing research and will update the air quality criteria accordingly. These revised criteria should provide a more informed basis for reaching a decision on whether a revised NAAQS or other regulatory measures are needed in the future.

In the interim, the 1990 Clean Air Act Amendments (Pub. L. 101-549, 104 Stat. 2399 (1990)) require EPA to promulgate a number of control measures to reduce NO_x emissions from both mobile and stationary sources. These reductions are in addition to those required under title IV of the 1990 Amendments (Pub. L. 101-549, secs. 401-413, 104 Stat. 2399, 2584-2634 (1990)). Title IV, in conjunction with other titles of the Act, requires EPA to reduce nitrogen oxide emissions by approximately two million tons from 1980 emission levels. The reductions achieved through these EPA initiatives will provide additional protection against the potential acute and chronic effects associated with exposure to NO_x while EPA continues to generate and review additional information on the effects of oxides of nitrogen on public welfare and the environment. The EPA believes it is important to continue to recognize the benefit to the environment that can be achieved by further reducing NO_x emissions. Therefore, as part of this process, the EPA will integrate, to the extent appropriate, nitrogen deposition considerations when assessing new NO_x control strategies.

III. Miscellaneous

A. Executive Order 12866

Under Executive Order 12866, the Agency must determine whether a regulatory action is "significant" and, therefore, subject to Office of Management and Budget (OMB) review and the requirements of the Executive Order. The order defines "significant regulatory action" as one that may:

(1) Have an annual effect on the economy of \$100 million or more or adversely affect in a material way the economy, a sector of the economy,

productivity, competition, jobs, the environment, public health or safety, or State, local, or tribal governments or communities;

(2) create a serious inconsistency or otherwise interfere with an action taken or planned by another Agency;

(3) materially alter the budgetary impact of entitlements, grants, user fees, or loan programs or the rights and obligations or recipients thereof; or

(4) raise novel legal or policy issues arising out of legal mandates, the President's priorities, or the principles set forth in the Executive Order.

Although the EPA is not proposing any modification of the existing NO₂ NAAQS, the OMB has advised the EPA that this proposal should be construed as a "significant regulatory action" within the meaning of the Executive Order. Accordingly, this action was submitted to the OMB for review. Any changes made in response to OMB suggestions or recommendations will be documented in the public record.

B. Regulatory Flexibility Analysis

The Regulatory Flexibility Act (RFA) requires that all Federal agencies consider the impacts of final regulations on small entities, which are defined to be small businesses, small organizations, and small governmental jurisdictions (5 U.S.C. 601 *et seq.*). These requirements are inapplicable to rules or other administrative actions for which the EPA is not required by the Administrative Procedure Act (APA), 5 U.S.C. 551 *et seq.*, or other law to publish a notice of proposed rulemaking (5 U.S.C. 603(a), 604(a)). The EPA has elected to use notice and comment procedures in deciding whether to revise the NO₂ standards based on its assessment of the importance of the issues. Under section 307(d) of the Act, as the EPA interprets it, neither the APA nor the Act requires rulemaking procedures where the Agency decides to retain existing NAAQS without change. Accordingly, the EPA has determined that the impact assessment requirements of the RFA are inapplicable to the decision proposed in this notice.

C. Impact on Reporting Requirements

There are no reporting requirements directly associated with an ambient air quality standard promulgated under section 109 of the Act (42 U.S.C. 7400). There are, however, reporting requirements associated with related sections of the Act, particularly sections 107, 110, 160, and 317 (42 U.S.C. 7407, 7410, 7460, and 7617). This proposal will not result in any changes in these reporting requirements since it would retain the existing level and averaging

times for both the primary and secondary standards.

D. Unfunded Mandates Reform Act

Title II of the Unfunded Mandates Reform Act of 1995 (UMRA), P.L. 104-4, establishes requirements for Federal agencies to assess the effects of their regulatory actions on State, local, and tribal governments and the private sector. Under section 202 of the UMRA, EPA generally must prepare a written statement, including a cost-benefit analysis, for proposed and final rules with "Federal mandates" that may result in expenditures to State, local and tribal governments, in the aggregate, or to the private sector, of \$100 million or more in any 1 year. Before promulgating an EPA rule for which a written statement is needed, section 205 of the UMRA generally requires EPA to identify and consider a reasonable number of regulatory alternatives and adopt the least costly, most cost-effective or least burdensome alternative that achieves the objectives of the rule. The provisions of section 205 do not apply when they are inconsistent with applicable law.

Before EPA establishes any regulatory requirements that may significantly or uniquely affect small governments, including tribal governments, it must have developed, under section 203 of the UMRA, a small government agency plan. The plan must provide for notifying potentially affected small governments, enabling officials of affected small governments to have meaningful and significant Federal intergovernmental mandates, and informing, educating, and advising small governments on compliance with the regulatory requirements.

A decision by the Administrator pursuant to section 109(d) of the Act not to propose any revision of the existing national primary and secondary standards for NO₂ does not require rulemaking procedures, and EPA has elected to provide notice and an opportunity for comment concerning this proposed decision in view of the importance of the issues. If the Administrator makes a final decision not to modify the existing NAAQS for NO₂, this will not impose any new expenditures on governments or on the private sector, or establish any new regulatory requirements affecting small governments. Accordingly, the EPA has determined that the provisions of sections 202, 203, and 205 of the UMRA do not apply to this proposed decision.

List of Subjects in 40 CFR Part 50

Environmental protection, Air pollution control, Carbon monoxide,

Lead, Nitrogen dioxide, Ozone,
Particulate matter, Sulfur oxides.

Dated: October 2, 1995.

Carol M. Browner,
Administrator.

References

- (1) Ahmed, T.; Dougherty, R.; Sackner, M. A. (1983a) Effect of NO₂ exposure on specific bronchial reactivity in subjects with allergic bronchial asthma [final report]. Warren, MI: General Motors Research Laboratories; contract report no. CR-83/07/BI.
- (2) Ahmed, T.; Dougherty, R.; Sackner, M. A. (1983b) Effect of 0.1 ppm NO₂ on pulmonary functions and non-specific bronchial reactivity of normals and asthmatics [final report]. Warren, MI: General Motors Research Laboratories; contract report no. CR-83/11/BI.
- (3) "American Petroleum Institute v. Costle," 665 F. 2d 1176 (D.C. Cir. 1981), cert. den. 102 S. Ct. 1737 (1982).
- (4) Aranyi, C.; Fenters, J.; Ehrlich, R.; Gardner, D. (1976) Scanning electron microscopy of alveolar macrophages after exposure to oxygen, nitrogen dioxide, and ozone. *Environ. Health Perspect.* 16: 180.
- (5) Avol, E. L.; Linn, W. S.; Venet, T. G.; Hackney, J. D. (1986) Short-term health-related effects of air pollution relatable to power plants: a combined laboratory and field study [final report: year 1]. Downey, CA: Rancho Los Amigos Medical Center, Environmental Health Service; R&D series 86-RD-75.
- (6) Avol, E. L.; Linn, W. S.; Peng, R. C.; Valencia, G.; Little, D.; Hackney, J. D. (1988) Laboratory study of asthmatic volunteers exposed to nitrogen dioxide and to ambient air pollution. *Am. Ind. Hyg. Assoc. J.* 49: 143-149.
- (7) Bauer, M. A.; Utell, M. J.; Morrow, P. E.; Speers, D. M.; Gibb, F. R. (1986) Inhalation of 0.30 ppm nitrogen dioxide potentiates exercise-induced bronchospasm in asthmatics. *Am. Rev. Respir. Dis.* 134: 1203-1208.
- (8) Brunekreef, B.; Dockery, D. W.; Speizer, F. E.; Ware, J. H.; Spengler, J. D.; Ferris, B. G. (1989) Home dampness and respiratory morbidity in children. *Am. Rev. Respir. Dis.* 140: 1,363-1,367.
- (9) Bylin, G.; Lindvall, T.; Rehn, T.; Sundin, B. (1985) Effects of short-term exposure to ambient nitrogen dioxide concentrations on human bronchial reactivity and lung function. *Eur. J. Respir. Dis.* 66: 205-217.
- (10) Chang, L. Y.; Graham, J. A.; Miller, F. J.; Ospital, J. J.; Crapo, J. D. (1986) Effects of subchronic inhalation of low concentrations of nitrogen dioxide. I. The proximal alveolar region of juvenile and adult rats. *Toxicol. Appl. Pharmacol.* 83: 46-61.
- (11) Coffin, D. L.; Gardner, D. E.; Sidorenko, G. I.; Pinigin, M. A. (1977) Role of time as a factor in the toxicity of chemical compounds in intermittent and continuous exposures. Part II. Effects of intermittent exposure. *J. Toxicol. Environ. Health* 3: 821-828.
- (12) D'Elia, C. F.; Sanders, J. G.; Boynton, W. R. (1986) Nutrient enrichment studies in a coastal plain estuary: phytoplankton growth in large-scale, continuous cultures. *Can. J. Fish. Aquat. Sci.* 43: 397-406.
- (13) D'Elia, C. F.; Taft, J.; Smullen, J. T.; Macknis, J. (1982) Nutrient enrichment. In: Chesapeake Bay Program technical studies: a synthesis. Annapolis, MD: U.S. Environmental Protection Agency; pp. 36-102. Available from: NTIS, Springfield, VA; PB84-111202.
- (14) De Temmerman L.; Ronse, A.; an den Cruys, K.; Meeus-Verdinne, K. (1988) Ammonia and pine tree dieback in Belgium. In: Mathy, P., ed. Air pollution and ecosystems: proceedings of an international symposium; May 1987; Grenoble, France. Boston, MA: D. Reidel Publishing Company; pp. 774-779.
- (15) Dijkstra, L.; Houthuijs, D.; Brunekreef, B.; Akkerman, I.; Boleij, J. S. M. (1990) Respiratory health effects of the indoor environment in a population of Dutch children. *Am. Rev. Respir. Dis.* 142: 1172-1178.
- (16) Dockery, D. W.; Spengler, J. D.; Neas, L. M.; Speizer, F. E.; Ferris, B. G., Jr.; Ware, J. H.; Brunekreef, B. (1989a) An epidemiologic study of respiratory health status and indicators of indoor air pollution from combustion sources. In: Harper, J. P., ed. Combustion processes and the quality of the indoor environment: transactions of an international specialty conference; September 1988; Niagara Falls, NY. Pittsburgh, PA: Air & Waste Management Association; pp. 262-271. (A&WMA transactions series:TR-15).
- (17) Driscoll, C. T.; Schaefer, D. A.; Molot, L. A.; Dillon, P. J. (1989) Summary of North American data. In: Malanchuk, J. L.; Nilsson, J., eds. The role of nitrogen in the acidification of soils and surface waters. Gotab, Sweden: Nordic Council of Ministers; pp. 6-1-6-45.
- (18) Driscoll, C. T.; Schaefer, D. A. (1989) Background on nitrogen processes. In: Malanchuk, J. L.; Nilsson, J., eds. The role of nitrogen in the acidification of soils and surface waters. Miljorapport, Sweden: Nordic Council of Ministers; pp. 4-1-4-12.
- (19) Driscoll, C. T.; Van Dreason, R. (1993) Seasonal and long-term temporal patterns in the chemistry of Adirondack lakes. *Water Air Soil Pollut.* 67: 319-344.
- (20) Ehrlich, R.; Henry, M. C. (1968) Chronic toxicity of nitrogen dioxide: I. effect on resistance to bacterial pneumonia. *Arch. Environ. Health* 17: 860-865.
- (21) Ehrlich, R.; Findlay, J. C.; Fenters, J. D.; Gardner, D. E. (1977) Health effects of short-term inhalation of nitrogen dioxide and ozone mixtures. *Environ. Res.* 14: 223-231.
- (22) Ekwo, E. E.; Weinberger, M. M.; Lachenbruch, P. A.; Huntley, W. H. (1983) Relationship of parental smoking and gas cooking to respiratory disease in children. *Chest* 84: 662-668.
- (23) Ferguson, P.; Robinson, R. N.; Press, M. C.; Lee, J. A. (1984) Element concentrations in five *Sphagnum* species in relation to atmospheric pollution. *J. Bryol.* 13: 107-114.
- (24) Fisher, T. R.; Harding, L. W., Jr.; Stanley, D. W.; Ward, L. G. (1988a) Phytoplankton, nutrients, and turbidity in the Chesapeake, Delaware, and Hudson estuaries. *Estuarine Coastal Shelf Sci.* 27: 61-93.
- (25) Folinsbee, L. J. (1992) Does nitrogen dioxide exposure increase airways responsiveness? *Toxicol. Ind. Health* 8: 1-11.
- (26) Friedlander, Sheldon K., Chairman, clean air Scientific Advisory Committee (CASAC), "Memorandum to EPA Administrator, Subject: CASAC Review of the Air Quality Criteria Document for Nitrogen Dioxides." June 19, 1981.
- (27) Friedlander, Sheldon K., Chairman, Clean Air Scientific Advisory Committee (CASAC), "Memorandum to EPA Administrator, Subject: CASAC Review and Closure of the OAQPS Staff Paper for Nitrogen Oxides." July 6, 1982.
- (28) Fujimaki, H.; Shimizu, F.; Kubota, K. (1982) Effect of subacute exposure to NO₂ on lymphocytes required for antibody responses. *Environ. Res.* 29: 280-286.
- (29) Gardner, D. E.; Coffin, D. L.; Pinigin, M. A.; Sidorenko, G. I. (1977a) Role of time as a factor in the toxicity of chemical compounds in intermittent and continuous exposures. Part I. Effects of continuous exposure. *J. Toxicol. Environ. Health* 3: 811-820.
- (30) Gardner, D. E.; Miller, F. J.; Blommer, E. J.; Coffin, D. L. (1977b) Relationships between nitrogen dioxide concentration, time, and level of effect using an animal infectivity model. In: Dimitriadis, B., ed. International conference on photochemical oxidant pollution and its control: proceedings, v. I; September 1976; Raleigh, NC. Research Triangle Park, NC: U.S. Environmental Protection Agency, Environmental Sciences Research Laboratory; pp. 513-525; EPA report no. EPA-600/3-77-001a. Available from: NTIS, Springfield, VA; PB-264232. (Ecological research series).
- (31) Gardner, D. E.; Miller, F. J.; Blommer, E. J.; Coffin, D. L. (1979) Influence of exposure mode on the toxicity of NO₂. *Environ. Health Perspect.* 30: 23-29.
- (32) Gardner, D. E.; Graham, J. A.; Illing, J. W.; Blommer, E. J.; Miller, F. J. (1980) Impact of exposure patterns on the toxicological response to NO₂ and modifications by added stressors. In: Proceedings of the US-USSR third joint symposium on problems of environmental health; October 1979; Suzdal, USSR. Research Triangle Park, NC: National Institute of Environmental Health Sciences; pp. 17-40.

- (33) Gardner, D. E.; Miller, F. J.; Illing, J. W.; Graham, J. A. (1982) Non-respiratory function of the lungs: host defenses against infection. In: Schneider, T.; Grant, L., eds. Air pollution by nitrogen oxides: proceedings of the US-Dutch international symposium; May; Maastricht, The Netherlands. Amsterdam, The Netherlands: Elsevier Scientific Publishing Company; pp. 401-415. (Studies in environmental science 21).
- (34) Glezen, W. P. (1989) Antecedents of chronic and recurrent lung disease: childhood respiratory trouble. *Am. Rev. Respir. Dis.* 140: 873-874.
- (35) Gold, D. R.; Tager, I. B.; Weiss, S. T.; Tosteson, T. D.; Speizer, F. E. (1989) Acute lower respiratory illness in childhood as a predictor of lung function and chronic respiratory symptoms. *Am. Rev. Respir. Dis.* 140: 877-884.
- (36) Goldstein, E.; Eagle, M. C.; Hoepflich, P. D. (1973) Effect of nitrogen dioxide on pulmonary bacterial defense mechanisms. *Arch. Environ. Health* 26: 202-204.
- (37) Graham, J. A.; Gardner, D. E.; Blommer, E. J.; House, D. E.; Menache, M. G.; Miller, F. J. (1987) Influence of exposure patterns of nitrogen dioxide and modifications by ozone on susceptibility to bacterial infectious disease in mice. *J. Toxicol. Environ. Health* 21: 113-125.
- (38) Hasselblad, V.; Eddy, D. M.; Kotchmar, D. J. (1992) Synthesis of environmental evidence: nitrogen dioxide epidemiology studies. *J. Air Waste Manage. Assoc.* 42: 662-671.
- (39) Hazucha, M. J.; Ginsberg, J. F.; McDonnell, W. F.; Haak, E. D., Jr.; Pimmel, R. L.; House, D. E.; Bromberg, P. A. (1982) Changes in bronchial reactivity of asthmatics and normals following exposures to 0.1 ppm NO₂. In: Schneider, T.; Grant, L., eds. Air pollution by nitrogen oxides: proceedings of the US-Dutch international symposium; May; Maastricht, The Netherlands. Amsterdam, The Netherlands: Elsevier Scientific Publishing Company; pp. 387-400. (Studies in environmental science 21).
- (40) Hazucha, M. J.; Ginsberg, J. F.; McDonnell, W. F.; Haak, E. D., Jr.; Pimmel, R. L.; Salaam, S. A.; House, D. E.; Bromberg, P. A. (1983) Effects of 0.1 ppm nitrogen dioxide on airways of normal and asthmatic subjects. *J. Appl. Physiol.: Respir. Environ. Exercise Physiol.* 54: 730-739.
- (41) Howes, B. L.; Dacey, J. W. H.; Goehring, D. D. (1986) Factors controlling the growth form of *Spartina alterniflora*: feedbacks between above-ground production, sediment oxidation, nitrogen and salinity. *J. Ecol.* 74: 881-898.
- (42) Husar, R. B. (1986) Emissions of sulfur dioxide and nitrogen oxides and trends for eastern North America. In: Acid deposition: long-term trends. Washington, DC: National Academy Press; pp. 48-92.
- (43) Ito, K. (1971) [Effect of nitrogen dioxide inhalation on influenza virus infection in mice]. *Nippon Eiseigaku Zasshi* 26: 304-314.
- (44) Jakab, G. J. (1987a) Modulation of pulmonary defense mechanisms by acute exposures to nitrogen dioxide. *Environ. Res.* 42: 215-228.
- (45) Jakab, G. J. (1987b) Modulation of pulmonary defense mechanisms by acute exposures to nitrogen dioxide. *Experientia Suppl.* 51: 235-242.
- (46) Jefferies, R. L.; Perkins, N. (1977) The effects on the vegetation of the additions of inorganic nutrients to salt marsh soils at Stiffkey, Norfolk. *J. Ecol.* 65: 867-882.
- (47) Johnson, D. W.; Lindberg, S. E., eds. (1992) Atmospheric deposition and forest nutrient cycling: a synthesis of the integrated forest study. Ecosystems. New York, NY: Springer-Verlag.
- (48) Kaufmann, P. R.; Herlihy, A. T.; Mitch, M. E.; Messer, J. J.; Overton, W. S. (1991) Stream chemistry in the eastern United States: 1. synoptic survey design, acid-base status, and regional patterns. *Water Resour. Res.* 27: 611-627.
- (49) Kerr, H. D.; Kulle, T. J.; McIlhenny, M. L.; Swidersky, P. (1979) Effects of nitrogen dioxide on pulmonary function in human subjects: an environmental chamber study. *Environ. Res.* 19: 392-404.
- (50) Klein, R. M.; Perkins, T. D. (1987) Cascades of causes and effects of forest decline. *Ambio* 16: 86-93.
- (51) Koenig, J. Q.; Covert, D. S.; Morgan, M. S.; Horiike, M.; Horiike, N.; Marshall, S. G.; Pierson, W. E. (1985) Acute effects of 0.12 ppm ozone or 0.12 ppm nitrogen dioxide on pulmonary function in healthy and asthmatic adolescents. *Am. Rev. Respir. Dis.* 132: 648-651.
- (52) Koenig, J. Q.; Pierson, W. E.; Marshall, S. G.; Covert, D. S.; Morgan, M. S.; Van Belle, G. (1987a) The effects of ozone and nitrogen dioxide on lung function in healthy and asthmatic adolescents. Cambridge, MA: Health Effects Institute; research report no. 14.
- (53) Koenig, J. Q.; Covert, D. S.; Marshall, S. G.; Van Belle, G.; Pierson, W. E. (1987b) The effects of ozone and nitrogen dioxide on pulmonary function in healthy and in asthmatic adolescents. *Am. Rev. Respir. Dis.* 136: 1152-1157.
- (54) "Lead Industries Association, Inc. v. EPA," 647 F. 2d 1130 (D.C. Cir. 1980), cert. den. 101 S. Ct. 621 (1980).
- (55) Lee, J. A.; Press, M. C.; Woodin, S. J. (1986) Effects of NO₂ on aquatic ecosystems. In: Environment and quality of life: study on the need for an NO₂ long-term limit value for the protection of terrestrial and aquatic ecosystems. Luxembourg: Commission of the European Communities; pp. 99-119.
- (56) Linn, W. S.; Solomon, J. C.; Trim, S. C.; Spier, C. E.; Shamoo, D. A.; Venet, T. G.; Avol, E. L.; Hackney, J. D. (1985b) Effects of exposure to 4 ppm nitrogen dioxide in healthy and asthmatic volunteers. *Arch. Environ. Health* 40: 234-239.
- (57) Linn, W. S.; Shamoo, D. A.; Avol, E. L.; Whynot, J. D.; Anderson, K. R.; Venet, T. G.; Hackney, J. D. (1986) Dose-response study of asthmatic volunteers exposed to nitrogen dioxide during intermittent exercise. *Arch. Environ. Health* 41: 292-296.
- (58) Lippmann, Morton (1984) CASAC Closure Letter to EPA Administrator William Ruckelshaus dated October 18, 1984.
- (59) Logofet, D. O.; Alexandrov, G. A. (1984) Modelling of matter cycle in a mesotrophic bog ecosystem: II. dynamic model and ecological succession. *Ecol. Modell.* 21: 259-276.
- (60) Margolis, H. A.; Waring, R. H. (1986) Carbon and nitrogen allocation patterns of Douglas-fir seedlings fertilized with nitrogen in autumn. II. Field performance. *Can. J. For. res.* 16: 903-909.
- (61) McComb, A. J.; Atkins, R. P.; Birch, P. B.; Gordon, D. M.; Lukateli, R. J. (1981) Eutrophication in the Peel-Harvey estuarine system, Western Australia. In: Neilson, B. J.; Cronin, L. E., eds. Estuaries and nutrients. Clifton, NJ: Humana Press; pp. 323-342.
- (62) McCurdy, T. R. (1994) Analysis of high 1 hour NO₂ values and associated annual averages using 1988-1992 data. Report of the Office of Air Quality Planning and Standards, Durham, NC. Available in Docket A-93-06.
- (63) Melia, R. J. W.; Florey, C. du V.; Altman, D. G.; Swan, A. V. (1977) Association between gas cooking and respiratory disease in children. *Br. Med. J.* 2: 149-152.
- (64) Melia, R. J. W.; Florey, C. du V.; Chinn, S. (1979) The relation between respiratory illness in primary schoolchildren and the use of gas for cooking: I—results from a national survey. *Int. J. Epidemiol.* 8: 333-338.
- (65) Melia, J.; Florey, C.; Sittampalam, Y.; Watkins, C. (1983) The relation between respiratory illness in infants and gas cooking in the UK: a preliminary report. In: Air quality 6th world congress: [proceedings of the International Union of Air Pollution Prevention Associations]; May; Paris, France. SEPIC (APPA); pp. 263-269.
- (66) Miller, F. J.; Graham, J. A.; Raub, J. A.; Illing, J. W.; Menache, M. G.; House, D. E.; Gardner, D. E. (1987) Evaluating the toxicity of urban patterns of oxidant gases. II. Effects in mice from chronic exposure to nitrogen dioxide. *J. Toxicol. Environ. Health* 21: 99-112.
- (67) Mochitate, K.; Takahashi, Y.; Ohsumi, T.; Miura, T. (1986) Activation and increment of alveolar macrophages induced by nitrogen dioxide. *J. Toxicol. Environ. Health* 17: 229-239.

- (68) Neas, L. M.; Ware, J. H.; Dockery, D. W.; Spengler, J. D.; Ferris, B. G., Jr.; Speizer, F. E. (1990) The association of indoor nitrogen dioxide levels with respiratory symptoms and pulmonary function in children. In: Indoor air '90: proceedings of the 5th international conference on indoor air quality and climate, volume 1, human health, comfort and performance; July-August; Toronto, ON, Canada. Ottawa, ON, Canada: International Conference on Indoor Air Quality and Climate, Inc.; pp. 381-386.
- (69) Neas, L. M.; Dockery, D. W.; Ware, J. H.; Spengler, J. D.; Speizer, F. E.; Ferris, B. G., Jr. (1991) Association of indoor nitrogen dioxide with respiratory symptoms and pulmonary function in children. *Am. J. Epidemiol.* 134: 204-219.
- (70) Neas, L. M.; Dockery, D. W.; Spengler, J. D.; Speizer, F. E.; Ferris, B. G., Jr. (1992) Variations in the association between indoor nitrogen dioxide and childhood respiratory symptoms by sampling location, season and source. *Am. Rev. Respir. Dis.* 145: A93.
- (71) Nixon, S. W.; Pilson, M. E. Q. (1983) Nitrogen in estuarine and coastal marine ecosystems. In: Carpenter, E. J.; Capone, D. G., eds. Nitrogen in the marine environment. New York, NY: Academic Press; pp. 565-648.
- (72) Ogston, S. A.; Florey, C. du V.; Walker, C. H. M. (1985) The Tayside infant morbidity and mortality study: effect on health of using gas for cooking. *Br. Med. J.* 290: 957-960.
- (73) "Oregon Natural Resource Council vs. EPA," No. 91-6529-HO (D. Or.) (1993).
- (74) Orehek, J.; Grimaldi, F.; Muls, E.; Durand, J. P.; Viala, A.; Charpin, J. (1981) Response bronchique aux allergenes apres exposition controlee au dioxyde d'azote [Bronchial response to allergens after controlled NO₂ exposure]. *Bull. Eur. Physiopathol. Respir.* 17: 911-915.
- (75) Parker, R. F.; Davis, J. K.; Cassell, G. H.; White, H.; Dziedzic, D.; Blalock, D. K.; Thorp, R. B.; Simecka, J. W. (1989) Short-term exposure to nitrogen dioxide enhances susceptibility to murine respiratory mycoplasmosis and decreases intrapulmonary killing of *Mycoplasma pulmonis*. *Am. Rev. Respir. Dis.* 140: 502-512.
- (76) Robison, T. W.; Duncan, D. P.; Forman, H. J. (1990) Chemoattractant and leukotriene B₄ production from rat alveolar macrophages exposed to nitrogen dioxide. *Am. J. Respir. Cell Mol. Biol.* 3: 21-26.
- (77) Rombout, P. J. A.; Dormans, J. A. M. A.; Marra, M.; Van Esch, G. J. (1986) Influence of exposure regimen on nitrogen dioxide-induced morphological changes in the rat lung. *Environ. Res.* 41: 466-480.
- (78) Samet, J. M.; Utell, M. J. (1990) The risk of nitrogen dioxide: what have we learned from epidemiological and clinical studies? *Toxicol. Ind. Health* 6: 247-262.
- (79) Samet, J. M.; Lambert, W. E.; Skipper, B. J.; Cushing, A. H.; Hunt, W. C.; Young, S. A.; McLaren, L. C.; Schwab, M.; Spengler, J. D. (1993) Health outcomes. In: Nitrogen dioxide and respiratory illness in children, part I. Cambridge, MA: Health Effects Institute; research report no. 58.
- (80) Schlesinger, R. B. (1987a) Effects of intermittent inhalation exposures to mixed atmospheres of NO₂ and H₂SO₄ on rabbit alveolar macrophages. *J. Toxicol. Environ. Health* 22: 301-312.
- (81) Schlesinger, R. B. (1987b) Intermittent inhalation of nitrogen dioxide: effects on rabbit alveolar macrophages. *J. Toxicol. Environ. Health* 21: 127-139.
- (82) Silsbee, D. G.; Larson, G. L. (1982) Water quality of streams in the Great Smoky Mountains National Park. *Hydrobiologia* 89: 97-115.
- (83) Simpson, J. C.; Olsen, A. R. (1990) Wet deposition temporal and spatial patterns in North America, 1987. Research Triangle Park, NC: U.S. Environmental Protection Agency, Atmospheric Research and Exposure Assessment Laboratory; EPA report no. EPA-600/4-90-019. Available from: NTIS, Springfield, VA; PB90-251836.
- (84) Smith, S. V. (1984) Phosphorus versus nitrogen limitation in the marine environment. *Limnol. Oceanogr.* 29: 1149-1160.
- (85) Smullen, J. T.; Taft, J. L.; Macknis, J. (1982) Nutrient and sediment loads to the tidal Chesapeake Bay system. In: Chesapeake Bay Program technical studies: a synthesis. Annapolis, MD: U.S. Environmental Protection Agency; pp. 150-251. Available from: NTIS, Springfield, VA; PB84-111202.
- (86) Suzuki, T.; Ikeda, S.; Kanoh, T.; Mizoguchi, I. (1986) Decreased phagocytosis and superoxide anion production in alveolar macrophages of rats exposed to nitrogen dioxide. *Arch. Environ. Contam. Toxicol.* 15: 733-739.
- (87) Tallis, J. H. (1964) Studies on southern Pennine peats: III. the behavior of *Sphagnum*. *J. Ecol.* 52: 345-353.
- (88) Tamm, C. O.; Popovic, B. (1974) Intensive fertilization with nitrogen as a stressing factor in a spruce ecosystem. I. Soil effects. Stockholm, Sweden: Royal College of Forestry. (Studia forestalia suecica nr. 121).
- (89) Tyler, M. (1988) Contribution of atmospheric nitrate deposition to nitrate loading in the Chesapeake Bay. Annapolis, MD: Department of Natural Resources, Chesapeake Bay Research & Monitoring Division; report no. AD-88-7.
- (90) U.S. Bureau of the Census. (1991) Statistical abstract of the United States: 1991. 111th ed. Washington, DC: U.S. Bureau of the Census; pp. 111 and 123.
- (91) U.S. Department of Health and Human Services. (1990) Vital and health statistics: current estimates from the National Health Interview Survey, 1989. Hyattsville, MD: Public Health Service, National Center for Health Statistics; DHHS publication no. (PHS) 90-1504. (Series 10: data from the National Health Survey no. 176).
- (92) U.S. Environmental Protection Agency. (1971) Air quality criteria for nitrogen oxides. Washington, DC: U.S. Environmental Protection Agency, Air Pollution Control Office; EPA report no. AP-84. Available from: NTIS, Springfield, VA; PB-197333/BE.
- (93) U.S. Environmental Protection Agency. (1982) Air quality criteria for oxides of nitrogen. Research Triangle Park, NC: Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office; EPA report no. EPA-600/8-82-026. Available from: NTIS, Springfield, VA; PB83-131011.
- (94) U.S. Environmental Protection Agency. (1985) Ambient water quality criteria for ammonia-1984. Washington, DC: Criteria and Standards Division; EPA report no. EPA-440/5-85-001. Available from: NTIS, Springfield, VA; PB85-227114.
- (95) U.S. Environmental Protection Agency. (1982b) Review of the National Ambient Air Quality Standards for Nitrogen Oxides: Assessment of Scientific and Technical Information. OAQPS Staff Paper. Office of Air Quality Planning and Standards; EPA report no. EPA-450/5-82-002. Available from: NTIS, Springfield, VA.
- (96) U.S. Environmental Protection Agency. (1993a) Air quality criteria for oxides of nitrogen. Research Triangle Park, NC: Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office; EPA report no. EPA-600/8-91/049F. Available from: NTIS, Springfield, VA.
- (97) U.S. Environmental Protection Agency. (1995) Review of the National Ambient Air Quality Standards for Nitrogen Oxides: Assessment of Scientific and Technical Information. OAQPS Staff Paper. Office of Air Quality Planning and Standards; EPA report no. EPA-452/R-95-005.
- (98) U.S. Environmental Protection Agency. (1995) An SAB Report: Review of the Acid Deposition Standard Feasibility Study Report to Congress. Prepared by the Acid Deposition Effects Subcommittee of the Ecological Processes and Effects Committee; EPA report no. EPA-SAB-EPEC-95-019, September 1995.
- (99) Van Breemen, N.; Van Dijk, H. F. G. (1988) Ecosystem effects of atmospheric deposition of nitrogen in the Netherlands. In: Dempster, J. P.; Manning, W. J.; Skeffington, R. A., eds. Excess nitrogen deposition: [papers from the workshop]; September 1987; Leatherhead, Surrey, United Kingdom. *Environ. Pollut.* 54: 249-274.

- (100) Van Dijk, H. F. G.; De Louw, M. H. J.; Roelofs, J. G. M.; Verburg, J. J. (1990) Impact of artificial, ammonium-enriched rainwater on soils and young coniferous trees in a greenhouse. Part II—effects on the trees. *Environ. Pollut.* 63: 41–59.
- (101) Van Miegroet, H.; Cole, D. W. (1984) The impact of nitrification on soil acidification and cation leaching in red alder ecosystem. *J. Environ. Qual.* 13: 586–590.
- (102) Vermeer, J. G. (1986) The effect of nutrients on shoot biomass and species composition of wetland and hayfield communities. *Acta Oecol. Oecol. Plant.* 7: 31–41.
- (103) Ware, J. H.; Dockery, D. W.; Spiro, A., III; Speizer, F. E.; Ferris, B. G., Jr. (1984) Passive smoking, gas cooking, and respiratory health of children living in six cities. *Am. Rev. Respir. Dis.* 129: 366–374.
- (104) Waring, R. H. Pitman, G. B. (1985) Modifying lodgepole pine stands to change susceptibility to mountain pine beetle attack. *Ecology* 66: 889–897.
- (105) White, T. C. R. (1984) The abundance of invertebrate herbivores in relation to the availability of nitrogen in stressed food plants. *Oecologia* 63:423–425.
- (106) Wigington, P. J., Jr.; Davies, T. D.; Tranter, M.; Eshleman, K. (1990) Episodic acidification of surface waters due to acidic deposition. Washington, DC: National Acid Precipitation Assessment Program. (Acidic deposition: state of science and technology report 12).
- (107) Wolff, G. T. (1993) CASAC closure letter for the 1993 Criteria Document for Oxides of Nitrogen addressed to U.S. EPA Administrator Carol M. Browner dated September 30, 1993.
- (108) Wolff, G. T. (1995) CASAC closure letter for the 1995 OAQPS Staff Paper addressed to U.S. EPA Administrator Carol M. Browner dated August 22, 1995.
- (109) Yamamoto, I.; Takahashi, M. (1984) Ultrastructural observations of rat lung exposed to nitrogen dioxide for 7 months. *Kitasato Arch. Exp. Med.* 57: 57–65.

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40 CFR Part 60

[AD-FRL–5308–9]

Standards of Performance for New Stationary Sources: Volatile Organic Compound Emissions From the Synthetic Organic Chemical Manufacturing Industry Wastewater

AGENCY: Environmental Protection Agency (EPA).

ACTION: Supplemental notice to proposed rule.

SUMMARY: Today's proposal clarifies the application of the proposed new source performance standards (NSPS) for

volatile organic compound (VOC) emissions from the synthetic organic chemical manufacturing industry (SOCMI) wastewater sources to modifications of existing SOCMI process units. The SOCMI wastewater NSPS were proposed on September 12, 1994 (59 FR 46780) under authority of Section 111 of the Clean Air Act, based on the Administrator's determination that VOC emissions from SOCMI wastewater operations cause, or contribute significantly to, air pollution that may reasonably be anticipated to endanger public health or welfare.

DATES: Comments on today's proposal must be received on or before November 13, 1995.

ADDRESSES: Interested parties may submit written comments regarding the amendments to the proposed rule (in duplicate if possible) to: Air and Radiation Docket and Information Center (6102), Attention, Docket No. A–94–32, U. S. Environmental Protection Agency, 401 M Street, SW, Washington, DC 20460. The EPA requests that a separate copy also be sent to Robert Lucas at the address listed below.

FOR FURTHER INFORMATION CONTACT: Mr. Robert Lucas at telephone (919) 541–0884, Emission Standards Division (MD–13), Office of Air Quality Planning and Standards, U. S. Environmental Protection Agency, Research Triangle Park, North Carolina 27711.

SUPPLEMENTARY INFORMATION: The amendments to the proposed regulatory text are not included in this **Federal Register** document, but are available in Docket No. A–94–32 or by request from the Air Docket (see **ADDRESSES**). This notice, the proposed regulatory text, the amendments to the proposed rule, and background information document are also available on the Technology Transfer Network (TTN), one of the EPA's electronic bulletin boards developed and operated by the Office of Air Quality Planning and Standards. The TTN provides information and technology exchange in various areas of air pollution control. The service is free, except for the cost of a phone call. Dial (919) 541–5742 for up to a 14,400 bits per second (bps) modem. If more information on the TTN is needed, call the HELP line at (919) 541–5384.

I. Background

On September 12, 1994, the EPA proposed standards to limit VOC emissions from SOCMI wastewater. The proposed standards would regulate VOC emissions from wastewater generated by SOCMI process units and are limited to emission points in the associated process unit's wastewater collection and

treatment system. The standards would require all new, modified, and reconstructed SOCMI process units to control wastewater emissions to the level achievable by the best demonstrated system of continuous emission reduction, considering costs, nonair quality health, and environmental and energy impacts. In addition to requiring end-of-pipe and add-on controls, the standards would also control VOC wastewater emissions by eliminating or reducing the formation of these pollutants.

Today's proposal clarifies how the SOCMI wastewater NSPS applies to modifications of existing SOCMI process units in response to concerns raised by representatives of the chemical manufacturing industry. The EPA is addressing some of the industry's concerns at this time, because modifications of SOCMI process units that generate wastewater that were modified after September 12, 1994, will be subject to the final NSPS. Additional issues raised by comments to the September 12, 1994 proposed rule will be addressed at the time that the final rule is promulgated.

II. Modification of Existing Process Units

a. Increased Emissions From Non-Wastewater Sources

Today's proposal clarifies that physical and operational changes to SOCMI process units that result in increased emissions from non-wastewater sources do not subject a process unit to the SOCMI wastewater NSPS. Under the existing regulatory framework any physical or operational change to a SOCMI process unit that results in an increase in emissions from any emission source within a process unit—irrespective of whether the increased emissions are from wastewater sources—could be considered to be a modification within the meaning of section 111 of the Act, 42 U.S.C. § 7411.¹ Accordingly, a physical or operational change to a SOCMI process unit that results in increased emissions from sources other than wastewater would subject an existing SOCMI process unit (that was

¹ The NSPS general provisions that address modifications provide that “. . . any physical or operational change to an existing facility which results in an increase in the emission rate to the atmosphere of any pollutant to which a standard applies shall be considered a modification within the meaning of section 111 of the Act. Upon modification, an existing facility shall become an affected facility for each pollutant to which a standard applies and for which there is an increase in the emission rate to the atmosphere.” (emphasis added)(40 CFR § 60.14(a))